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RATIONAL USE OF PESTICIDES

In selecting the subject matter for this journal, which completes its first volume with this issue, one of our aims has been to make it easy for the specialist to acquire a general knowledge of current work in fields other than his own. Most problems in agriculture call for contributions from all branches of science, but it is upon integration of the results and their application that the final solution depends, and this requires breadth of view from those who share in the work.

This need is evident, for example, in the study of the complex biological problems that have followed the use of pesticides. When such remarkable toxicants as the synthetic organic insecticides, growth-regulator herbicides, and the antibiotics first appeared it seemed that these would end many of the troubles that afflict crops and farm animals. They have indeed proved to be of immense value in preventing losses, but undesirable effects have occasionally resulted from their indiscriminate application, and means must be found to avoid them.

All entomologists are now aware of the ecological consequences of the use of the new insecticides. Apart from their destructive effects on various forms of wild life, which can be minimized by care in application, two phenomena have developed for which practical remedies have not yet been discovered. One is the increase in the pest populations after treatment, sometimes to a higher level than before—and this may apply not only to the pest it was hoped to control but to previously minor pests in the sprayed area, as instanced by the build-up of red spider mites. A number of chemicals are implicated, and it has been established that they produce these effects by destroying predators and parasites that normally help to keep certain pests in check. The situation affects only a small number of species of economic importance and arises chiefly in orchards where pesticides are regularly used. The second phenomenon, which is more serious, is the appearance and spread of insecticide-resistant strains among populations that were once susceptible, in much the same way as resistance to antibiotics has developed in bacteria. Its most disturbing feature is that resistance is inherited and comes to stay.

Effects of these kinds seem to be less marked in other fields of plant protection. In weed control, for example, the destruction of natural enemies is of little practical importance owing to the inherent stability of weed populations, and a species is neither liable to be exterminated by normal application of herbicides nor to be replaced by a minor species resistant to the treatment. But the appearance of resistance in susceptible species has been recorded, and this danger cannot be ignored.

For these and other reasons more attention is now being paid to the "intelligent use" of pest control products. It is generally agreed that, where biotic upsets are likely to occur, we should not apply pesticides in the field as we would in the greenhouse or grain store but should take account of all organisms in the environment and disturb their natural balance as little as possible. How this can best be done, however, is a matter upon which opinions are divided.

To the ecologist the problem is one in population dynamics. He looks on the use of chemicals merely as one of many environmental factors having repercussions on the local fauna and flora, hence not to be considered alone. His approach to an outbreak, therefore, is to study all the factors involved before attempting to seek a remedy, and his objective is not the elimination of the noxious species but the creation of conditions under which its numbers will be kept down to the level at which it does the least possible harm, using chemical treatment only as a supplement to husbandry measures to achieve this. The concept is reasonable and may be the ideal way of dealing with certain entomological problems, as it is for internal parasites of grazing animals, but it is doubtful if methods can ever be devised that will be effective under the varying farming systems in which they have to operate.

The biochemist takes a realistic and more hopeful view of the situation. Indeed, he sees a practical answer to most of the problems described in the discovery of chemicals of greater specificity and other contrasting properties, and we may reasonably expect him to find them. In the meantime, much can be done with the materials now available by careful attention to dosage and time of application, and by such expedients as the use of unrelated chemicals in rotation or in conjunction. Since crops and stock must be saved, the rational course would appear to be to use the chemicals needed to protect them, in ways compatible with the principles of good husbandry. It will sometimes be advisable not to press the attack too far, but in the broad field of chemotherapy there are innumerable instances where crop protection products can be freely used and very few where they give rise to undesirable side effects.

THE MANURING OF SUGAR CANE

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Results of the world's fertilizer experiments on sugar cane, summarized in this paper, show that yields can be greatly increased in all countries and on all soil types by applying the right fertilizers in adequate amounts at the right times. Yet dressings are usually much less than the recommended amounts, especially on smallholdings, and there is still need for more research on many aspects of manuring even on progressive estates.

Probably more research has been devoted to sugar cane than to any other tropical crop. The literature on sugar cane is so voluminous and widely scattered that until the last few years, when a number of comprehensive works on the crop appeared [1, 5, 11, 20, 30, 33, 36], it has been difficult, even with the aid of abstract journals, to obtain a general picture of the latest information on cane growing throughout the world. The resumption in 1950 of the triennial congresses of the International Society of Sugar Cane Technologists, after an interval of twelve years, has stimulated afresh the international exchange of knowledge and experience.

Comparing methods of production in the various sugar cane growing countries one cannot but be impressed by the diversity of field practices and the wide margin that exists, in terms of raw sugar per acre, between the most efficient and the least efficient producers. Climate is undoubtedly a limiting factor. Growers outside the tropics are handicapped by the shortness of the active growing season and, in some places, by the need to finish harvesting before frost renders the cane unfit for milling. Elsewhere, as in some of the smaller islands in the West Indies, lack of rainfall may impose an upper limit to the yield, and in many parts of the world hurricanes are a frequent source of damage. The yield of raw sugar per acre is also governed by non-agricultural factors, such as speed of delivery of the cane to the mills and milling efficiency. In many parts of the Far East the density of population is such that sugar cane must be grown in rotation with rice and other food crops, and planting and harvesting of the cane may have to be advanced or delayed accordingly. Where pressure on the land is so high, the farmer can rarely afford to practise ratooning. In Java, for example, it is possible to take two crops of rice and one of maize in the time it takes to grow a single crop of sugar cane. In less densely populated countries where there is no shortage of fertile land it may sometimes pay to grow cane continuously, taking many ratoon crops and moving whenever necessary on to new land; but, where the supply of land is limited, monoculture is possible only if special steps are taken to conserve soil fertility by the use of fertilizers, green manuring, flood-fallowing, etc.

In the past fifty years the world production of raw cane sugar has trebled, while the acreage under cane has probably not more than doubled. A remarkable feature of this expansion has been the success of the plant breeder in raising disease-resistant and high-yielding varieties. More efficient control of water has also played an important part in raising the level of production, e.g. by extension of irrigation in drier districts, using trash as a mulch to conserve soil moisture instead of burning and, in the wetter districts, by effective drainage and planting on raised and cambered beds. The present paper is concerned with a third aspect—knowledge of the crop's nutrient requirements and means of satisfying these by manuring.

NUTRITIONAL REQUIREMENTS

A crop of sugar cane yielding 40 tons of millable stalks and 24 tons of tops and trash to the acre may be reckoned to contain: in the stalks, 45 lb. N, 45 lb. P_2O_5 and 145 lb. K_2O*; in the tops and trash, 90 : 45 : 125; and in the underground portions, 45 : 20 : 20. These figures give a general indication of the needs that have to be met from fertilizers and from the soil, after making allowance for residues, such as molasses or filter mud, that may be returned. For the purpose of calculation it may be assumed that, after the cane has been milled, about three-quarters of the potassium originally present in the stalks is concentrated in the molasses, and one-third or more of the phosphorus is precipitated in the filter mud.

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* Such proportions are subsequently expressed in ratio form, e.g. 45 : 45 : 145.

Response to Fertilizers

The most comprehensive analyses of fertilizer experiments on sugar cane in British Colonial and Commonwealth territories are those of Carey and Robinson [6] and of Hodnett [21]. The first of these summarizes the results of about 1500 experiments conducted between 1900 and 1943. The second and more recent survey is restricted to experiments of modern randomized design and gives the results of just over 1000 experiments conducted since 1930, rather more than half of which were not included in the earlier summary.

By assuming response curves of the form: $y = y_0 + \alpha(1 - 10^{-kx})$, where y is the yield per acre with a dressing of x lb. per acre, y_0 is the yield with no fertilizer, α is the limiting response, and k is a positive constant which determines the curvature of the response curve, these authors were able to convert the results of all experiments into responses to standard dressings of 50 : 50 : 50 (Table I). The following values of k were adopted:

	k (acres/lb.)		
	N	P_2O_5	K_2O
Cane	0·006	0·010	0·010
Sugar	0·008	0·010	0·008

TABLE I

MEAN RESPONSES OF CANE (TON/ACRE) TO STANDARD DRESSINGS OF 50 LB. N, 50 LB. P_2O_5 AND 50 LB. K_2O PER ACRE [6]

Territory	N		P_2O_5	K_2O
	Plant cane	Ratoons		
Jamaica ..	2·1	2·3	0·0	1·6
Leeward Is. ..	2·2	4·5	−0·1	1·3
Barbados ..	1·9	5·0	0·1	1·0
Trinidad ..	2·8	5·3	2·3	2·4
British Guiana	5·0	5·7	2·1	0·8
South Africa ..	0·9	3·3	4·3	2·5
Mauritius ..	1·6	2·8	3·9	2·7
India	4·2		1·5	0·3
Queensland ..	2·4	6·0	1·9	1·8

In order to derive standard responses in terms of sugar per acre from the largest possible number of experiments, Hodnett first calculated the average effects of standard fertilizer dressings on the percentage of sugar in the cane from the experiments where this information was available and then applied these results to the other experiments where only the cane yields had been recorded (Table II).

In interpreting these tables it must be borne in mind that, although Hodnett followed Carey and Robinson in adopting 50 lb./acre as the standard dressing for each plant nutrient, the average dressings in the experiments—and indeed the official recommendations in most of the countries concerned—are appreciably higher. Probably 100 lb./acre would be a better standard. Theoretical responses in cwt. sugar to 100 lb. N or K_2O per acre may be obtained quite simply by multiplying by 1·40 the responses to 50 lb./acre. The corresponding factor for P_2O_5 is 1·32.

Nitrogen is without question the most important fertilizer nutrient for sugar cane. Responses to nitrogen were observed in all the territories and on all soil types, but were less marked in South Africa, St. Kitts, Antigua, and on the lateritic soils in Jamaica where the average rainfall was low (below 40 in. per annum). Because irrigation increased the response to N, even where the rainfall was relatively high, it was evident that the seasonal distribution of the supply of moisture was almost as important as the total quantity received. Ammonium sulphate usually gave slightly higher yields of sugar than ammonium nitrate, sodium nitrate or calcium cyanamide at equivalent rates of N. There was no conclusive evidence of varietal differences in response.

Responses to phosphorus were mostly small except on certain soil types. Substantial residual responses of first ratoons to phosphatic fertilizer applied to plant cane were recorded, particularly on those soils where the plant-cane response was large.

Responses to potassium were also found to be associated with soil type and the results of soil analysis, but the residual effects were small.

Recommended Fertilizer Dressings

The Centre d'Etude de l'Azote's Bulletin [20], which was completed before Hodnett's survey had appeared, deals with nearly all major producers, outlines current practices, and reviews the results of fertilizer experiments, country by country. The recommended rates of N fertilizer application are summarized in Table III.

Generally speaking, the requirement of N per acre for unirrigated cane in districts with an

TABLE II

MEAN RESPONSES IN SUGAR YIELD (CWT./ACRE) TO STANDARD DRESSINGS OF 50 LB. N, 50 LB. P_2O_5 AND 50 LB. K_2O PER ACRE [21]

Territory	N		P_2O_5		K_2O	
	Plant cane	1st ratoon	Plant cane	1st ratoon	Plant cane	1st ratoon
Jamaica:						
Irrigated	10·5	13·2	2·2	2·5	5·5	7·7
Unirrigated	7·1	10·1				
Laterite unirrigated	3·9	4·5				
St. Kitts	3·5	22·8	3·0	4·0	5·0	5·2
Antigua	2·5	10·1	2·0	2·4	1·6	0·9
Barbados	6·5–10·6*	14·8–18·5*	0·4	3·0	1·6–6·2*	6·4–7·7*
Trinidad	10·8	8·8	3·9	3·3	5·0	4·7
British Guiana:						
Frontland:						
Unflooded	11·4	9·5	1·3	1·1	3·4	2·5
Flooded	6·2	17·5				
Pegasse	8·0	6·3	4·6	5·9		
South Africa	3·1	7·8	2·4–9·3*	2·7–6·8*	2·2–6·8*	2·5–11·5*
Mauritius:						
Irrigated	6·6	18·4	0·1	0·3	2·1	2·5
Unirrigated	3·8	10·5				
Queensland	5·6†	15·6	4·5–8·6*	2·1–8·2*	3·2–9·2*	2·8–8·0*

* According to soil type. † Response in Burdekin area was 20·6.

annual rainfall of 40–50 in. is about 40–60 lb. for plant cane (except after ploughing in a green manure) and 40–80 lb. for ratoons. In districts with 60–100 in. rainfall 100 lb. or more may be used effectively, particularly on ratoons; and with irrigation the total may be raised to 200–300 lb. given in several dressings.

The amounts of fertilizer applied vary considerably from country to country. Cuba and the Dominican Republic, for example, provide an interesting comparison with Puerto Rico and other islands in the West Indies. Sugar cane has long been the dominant crop in Cuba, and in the peak year of 1951–52 its exports accounted for over 40% of all sugar, cane and beet, entering international trade. Because of the long tradition of continuous and exhaustive cropping, fluctuating prices and crop limitation schemes, there has been little incentive until recently either to introduce improved varieties or to use fertilizers, except on certain limited areas. Although experiments on various soil types have indicated an optimum of perhaps 60 : 60 : 40, the average use in 1949–55 was less than one-third of this amount. Similarly in the Dominican Republic, where recent experiments have demonstrated that 30–60 lb. N/acre may be used to advantage on unirrigated cane and as much as 250 lb. with irrigation, the average use over the same seven-year period was no more than 12 lb. In marked contrast the adjacent island of Puerto Rico uses an average of 160 : 45 : 110 [3]. In Jamaica [26] in 1951 nearly all the estate cane area received an average of 76 lb. N/acre; but only 8% was treated with phosphate (av. 37 lb. P_2O_5/acre) and 35% with potassium (av. 96 lb. K_2O/acre).

In Latin America and Southern Asia the contrast between fertilizer practices on progressive and backward estates is particularly marked; many small planters use little or no fertilizer.

Spain, though only a minor producer, deserves special mention for the high yields obtained, further from the equator than any other cane-growing area, with a normal application of about 200 : 150 : 100 [23].

TABLE III

RECOMMENDED DRESSINGS OF NITROGEN FERTILIZER IN THE PRINCIPAL CANE-GROWING AREAS

Territory	Nitrogen (lb./acre)	Ref.
Africa:		
Egypt	Plant cane, 60–110; ratoons, up to 230	20
Madagascar	55–90	14, 38
Mauritius	Plant cane, 60; ratoons, 125	20
Natal	Plant cane, 20; ratoons, 90–120	
Réunion	Similar to Mauritius	17
India:		
India*:		
North	Irrigated, 80–150; unirrigated, 40–90	20
South	Irrigated, up to 300 (⅔ oil cake)	20
W. Pakistan*	Irrigated, 100	20
Far East:		
Formosa	135–270	41
Java	95	20
Philippines* (Negros)	90–110	20
Australia:		
Queensland	Plant cane, 35–65; ratoons, 45–110	20
Central and N. America:		
Hawaiian Is.	Plant cane, 200; ratoons, 165	20
Louisiana	Plant cane, 40–80; ratoons, 60–100	20
Mexico	100	20
West Indies:		
Barbados	As for Jamaica	20
Cuba*	60	20
Dominican Republic	Irrigated, up to 250; unirrigated, 30–60	20
Guadeloupe	Somewhat less than Martinique	7
Jamaica	Plant cane, 45–90; ratoons, 45–140	20
Leeward Is.	As for Jamaica	20
Martinique	60–220	2
Puerto Rico	125–250	20
Trinidad	Plant cane, 90–140; ratoons, 70–90	20
South America:		
Argentina (Tucumán)	50–70	20
Brazil*:		20
Minas Gerais	40–85	
Pernambuco	50–110	
Rio de Janeiro	40–85	
São Paulo	20–45	
British Guiana	Plant cane after flood fallow, 45; other, 90	20
Colombia	Nil	35
Peru	180 (⅔ guano)	20
Venezuela*	Plant cane, 50–110; ratoons, 80–160	12, 16

* Current use is much less than that recommended.

South Africa was formerly exceptional in that phosphorous was regarded as the major requirement, but recent experience with trash-mulched cane indicates that at least 90–120 lb. N/acre should be used on all ratoons and that a smaller dressing on plant cane may prove advantageous under certain conditions. Application of potassium fertilizer on sugar cane in South Africa increased nearly sixfold between 1951 and 1955, and the average recommendation is now 75–100 lb. K_2O/acre. Except on virgin soils, which still need large amounts of phosphorus fertilizer, the corresponding phosphorus requirement is about 50 lb. P_2O_5/acre applied in the furrow to plant cane and on top of the trash to ratoons.

In Florida, potassium is the major need, and on some highly organic soils it is usual to apply 50–100 lb. K_2O/acre, with micronutrients where necessary.

ESTIMATION OF NUTRIENT REQUIREMENTS

Soil Analysis

In a number of countries soil analysis is used as a rough guide to the need for phosphatic and potassic fertilizers. In South Africa, for example, a soil containing less than 40 p.p.m. P_2O_5 soluble in 1% citric acid (Dyer) and 0·15 m.e. exch. K/100 g. would be regarded as low in these nutrients (15). Australian soils give similar indications (29). In Mauritius 33 p.p.m. available P_2O_5 (Dyer) is about the threshold in soils with under 60 in. annual rainfall (10). In Trinidad, according to Hodnett (22), there appears to be decidedly more response to phosphatic fertilizers on soils which analyse less than 30 p.p.m. P_2O_5 (Truog), and Turner [39] recommends that potash should be applied to sugar cane soils with less than 90 p.p.m. exchangeable K_2O (0·19 m.e. exch. K/100 g.). The corresponding figure for St. Kitts is 130 p.p.m. exchangeable K_2O [40]. Similarly in Hawaii a response to potash is expected only where the exchangeable potassium in the soil is below 75 p.p.m. at the time of planting.

Foliar Diagnosis

A more favoured method of estimating nutrient requirements is that of leaf analysis or foliar diagnosis, which was first applied to sugar cane in Mauritius in about 1936 and has since been investigated and developed in many other countries. Normal practice in Mauritius [19] is to select about 60 third-leaf blades (counting the one partially unfolded as the first) from the primary shoots of ratoon cane aged 5–7 months. Chemical analyses are based on discs not exceeding 0·2 in. in diameter punched from the central portion of the leaf blades, excluding the midribs. A composition of 0·45–0·55% P_2O_5 and 1·35–1·65% K_2O (based on dry matter) is regarded as satisfactory; only below these levels would a response to P and K be expected.

In Jamaica, where the third *fully opened* leaf is selected, Innes [27] has concluded that no response to K may be expected if the leaf potash is over 1·4%, nor to P if the leaf phosphate exceeds 0·5%; satisfactory levels for leaf nitrogen at 5 months are 2·3% for plant cane and, for ratoons, 1·83%. A practical application of this method for N is to give an initial dressing of, say, 4 cwt. ammonium sulphate per acre, and to base the subsequent dressing on the result of foliar diagnosis. It has also been found that the relative response in leaf N at 4 months or even earlier—i.e. the difference in leaf N between plants with and without the initial N dressing—can give an accurate indication of the final response in yield of cane. A similar differential technique is applicable to P and K.

In British Guiana the leaf used for analysis is that with the top visible blade-joint, and the lowest satisfactory nutrient levels, expressed as percentages in the leaf dry matter, are 0·48% P_2O_5, 1·44% K_2O and, for 4½-month-old plant cane, about 2·2% N and, for ratoons, 1·7% N [18].

For nitrogen, Burr [4] in Hawaii prefers the alcohol-soluble nitrogen fraction of the stalk as an index. Evans [18], however, has found that the best results are obtained by analysing both the leaf and the brei or sawdust obtained by sawing through the three basal internodes. Tentative values for a satisfactory level of nitrogen in the brei, based on the limited information available, are 0·55–0·60% at 4½ months.

In Puerto Rico, where the leaves selected for analysis are the four nearest to the last-emerged unopened leaf, it has been found [37] that for every 10 in. of rain falling in the three months between leaf-sampling and harvesting there is an increase of about 0·2% N in the dry matter; after adjusting to a standard rainfall of 20 in. in this period the critical level of nitrogen is about 1·4%. Critical levels for other nutrients are about 0·17% P_2O_5 and 1·7% K_2O [31].

An elaboration of foliar diagnosis has been developed in the Hawaiian Islands under the name of "crop logging" [8, 9]. The principle of this system is that a continuous graphical record is kept of the nutrient status, weather conditions and fertilizer and irrigation treatments throughout the life of a crop. Sampling begins when the cane is 2–3 months old and continues at intervals of 35 days until harvest. The leaves used are the third to the sixth, counting from the small central leaf at the terminal bud. Only the leaf sheaths are used for assessing P and K and the central part of the leaf blade for N. Fertilizer and irrigation treatments are decided on the basis of the current log, studied in conjunction with the logs of previous crops on the same field.

The chief limitations of foliar diagnosis are that knowledge of nutrient deficiencies may come too late for satisfactory corrective measures to be taken, and that the method ceases to be applicable under exceptional conditions of drought, strong wind, rainfall, or incidence of pests and diseases. Nevertheless it does indicate areas that need further study, though a background of field experiments to which to key its results is necessary.

FERTILIZERS AND THEIR APPLICATION

Timing of Applications

To obtain maximum production sugar cane must have adequate supplies of plant nutrients throughout its life, particularly during the boom stage of growth. Compared with other nutrients, N is taken up more rapidly in the earlier months and is more readily leached from the soil. It is therefore usual to apply a part of the nitrogen fertilizer either at the time of planting or ratooning or a few weeks later, depending on climate and local conditions, and to apply the remainder in one or more top dressings during the active period of growth, co-ordinated with other agricultural operations where possible. Except where fertilizers can be applied in the irrigation water or from aircraft, it is necessary to give the final dressing before the crop has closed in. Application in the irrigation water tends to result in uneven distribution, for plants near the irrigation source receive considerably more fertilizer than those further away; the loss of nitrogen is about 20% per 200-foot run, according to experience in Hawaii [25]. The alternative of spraying concentrated solutions of urea and other soluble fertilizers from the air does not have this disadvantage. Application of solid materials from the air has also been tested [42]. Another method would be to use slow-acting nitrogenous fertilizers capable of making available a supply of nitrogen throughout the growing season, e.g. urea-formaldehyde products; but these are too costly for large-scale use.

Where needed, a single dressing of phosphatic fertilizer at or soon after planting usually suffices for the plant crop, and it may have an appreciable residual value for the succeeding ratoons. Studies in Hawaii [24] with radioactive phosphorus have shown that the best position for phosphatic fertilizers is immediately beneath the seed-piece. Placement 4–6 in. deep and 10–12 in. away from the centres of the stools, which is about as near as is possible with mechanical equipment, is nearly as good. The greatest advantage from placement occurs under irrigated conditions in dry districts where the soils are alternatively wet and dry and the cane roots penetrate deeply, as opposed to wet districts where the soils are continually moist and the roots grow near the surface, especially when under a blanket of trash.

The effect of K is generally less lasting than that of P, partly owing to luxury uptake; but, except where the growing season is very long, a single application at planting or ratooning is enough for one crop.

Types of Fertilizer

Ammonium sulphate (21% N) is still the most favoured nitrogenous fertilizer for sugar cane on account of its low cost and good physical condition; but in recent years there has been a growing interest in more concentrated fertilizers, particularly in liquid form.

Anhydrous ammonia (82% N) is a gas at ordinary temperatures but can be transported and stored under pressure as a liquid, and special tractor-mounted equipment for injecting it into the soil and covering to prevent loss has been developed. The ammonia may also be dissolved in irrigation water, usually at 50–70 p.p.m.

Aqueous ammonia (30% ammonia in water = 24·7% N, or 23% ammonia in water = 18·9% N) can be applied in the same way as the anhydrous product and has the advantage of not requiring high-pressure equipment—the pressure of the 30% solution at 50° C is only about 35 lb./sq. in. compared with nearly 300 lb./sq. in. for anhydrous ammonia, while that of the 23% solution is negligible. Although aqueous ammonia is already widely used in a number of countries, experience in Jamaica [28] suggests that it may be less effective per unit of N, when injected into the soil or dissolved in irrigation water, than ammonium sulphate broadcast.

Urea (up to 46% N), the most concentrated solid nitrogenous fertilizer, is highly soluble in water and has been successfully used in Hawaii for spraying sugar cane from the air. Field experiments to compare solid urea with ammonium sulphate were laid down in Mauritius in 1956, but the full results are not yet available. Preliminary results of pot experiments conducted in Mauritius seem to indicate that biuret, which is present in small quantities in fertilizer-grade urea, may be toxic to cane, but only in amounts far in excess of those likely to be employed in practice [34].

Other concentrated nitrogenous fertilizers include ammonium nitrate (35% N), urea-ammonia liquor and solutions of urea-ammonium nitrate and ammonia-ammonium nitrate (30–45% N).

The principal commercial potassic fertilizers used on sugar cane are potassium chloride (48–62% K_2O) and potassium sulphate (48–52% K_2O). The chief commercial source of phosphorus is superphosphate (14–21% water-soluble P_2O_5).

Micronutrients

Deficiencies of various micronutrients have been reported. Visual symptoms and foliar analysis are both useful in diagnosis. Manganese deficiency, encountered in Florida and Hawaii (where it is known as Pahala blight [32]), causes yellow-and-white striping of the leaf blades, especially towards the tips, and the leaves which are often twisted and intertwined may split or crack along the stripes. Copper deficiency, which occurs in parts of Florida, Queensland, Natal and possibly British Guiana, causes stunted, chlorotic plants, and the young leaves may fail to unroll. Lack of zinc is sometimes associated with that of copper. Molybdenum deficiency, reported from British Guiana, is characterized by the appearance on mature leaves of yellow streaks which later redden and form lesions. The symptoms of boron deficiency, observed in Hawaii, resemble the twisted top and ladder-like lesions of pokkah boeng disease, caused by the fungus *Gibberella fujikuroi.*

Deficiencies of any of these elements can be satisfactorily corrected by applying the appropriate micronutrient to the soil—for example, manganese sulphate at 65 lb./acre for manganese deficiency and copper sulphate at 25 lb./acre for copper deficiency—or by foliar sprays, or by injection into the stems of seed cane.

THE FUTURE

The greatest scope for the increased use of fertilizers on sugar cane is in countries where production is still largely in the hands of small growers, notably in Latin America and the Far East, and in areas where irrigation can be extended. On progressive estates there is likely to be more general use of diagnostic techniques to ensure that fertilizers are used to the best advantage; there will certainly be more research on the newer and more concentrated nitrogenous fertilizers, and timing and methods of application, such as machine-placement, solution in irrigation water and aerial spraying, will receive further attention. The extension of sugar cane growing into new areas, for example in East Africa, Ethiopia and the Sudan, calls for more basic experiments on the requirements of the crop under these conditions. In all cane-growing areas of the world there is need for more fundamental experiments on crop rotations and conservation practices designed to maintain fertility at a high level.

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HORMONES IN MEAT PRODUCTION

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A recent development of much interest to the livestock farmer is the use of synthetic oestrogens in the production of beef and mutton. Numerous trials have proved that such hormones, implanted under the skin or fed in concentrates, not only increase the rate of liveweight gain in cattle and sheep but improve carcass quality and the efficiency of food conversion. The practice is already common in the U.S.A. and is spreading in the U.K.

In dealing with factors that directly affect growth and fattening of farm animals it is usual to separate growth processes into two phases. These are: actual increase in size, mass and liveweight of the animal as a whole; and changes in the relative proportion and chemical composition of different parts of the body as the animal grows up.

The first phase is of particular interest to the farmer because most meat-producing animals are sold on a liveweight basis, and gain in weight depends to a large extent on the food consumption. In general, the greater the gain per day the greater is the efficiency of food conversion. Since more feed is needed to produce 1 lb. of body fat than 1 lb. of lean meat, conversion is improved when the animal puts on muscle rather than fat.

The second phase, changes in body proportions and composition, is mainly of concern to the butcher. It determines how much of the purchased live animal he has to sell as a carcass, whether the carcass will give a high proportion of the joints that are in greatest demand and so command higher prices, and whether the composition of these joints in terms of muscle fat and bone is such as will suit consumer requirements [16].

NATURAL AND SYNTHETIC HORMONES

Both the liveweight gain and body proportions of meat animals are well known to be affected by such things as breed, feed, age and sex, but until recently the effects of hormones have received little attention. Although various hormones influence the development of the body, only the oestrogenic hormones have so far been used under practical conditions for promoting growth. Their application to improve the flesh of poultry by caponization has been widely adopted for some time, but not until 1948 did use begin to be found for them in the production of beef and mutton. This article deals with the new development.

Hormones, like "organizer substances" in embryonic development, usually act on specific cells or sets of cells in tissues, causing them to develop and function at a higher rate and conferring upon them a priority over many other cells in the body for nutrients from the blood. The hormones are normally produced in different parts of the body in small glands which discharge their particular hormone into the bloodstream. The key organ in this respect is the anterior pituitary, a small gland at the base of the brain. This secretes several hormones which act on other glands so that these in turn produce other hormones; it is, for example, the source of "follicle-stimulating hormone" [FSH] which acts on the ovary to cause ripening of the follicles containing the egg, and these ripening follicles give rise to the oestrogenic hormones which act on the various parts of the body to promote the development of the female characters, such as the growth of the nipples. Whereas the hormones of the anterior pituitary gland are complex substances containing proteins as an integral part of their structure, and therefore most difficult to synthesize, many of the secondary hormones, including the oestrogens, have a comparatively simple structure and can be synthesized cheaply [1]. The two most frequently used synthetic oestrogens for growth purposes have been stilboestrol and hexoestrol.

Until recently the main hormone known to affect growth was the growth hormone of the anterior pituitary gland, best obtained from the ox. In 1933 Evans and others [8] showed that when growth hormone was injected into young dogs and other animals their rate of growth was greatly increased (Figure 1). Since then much work has been done with the substance. Mutations that produce dwarf forms in animals have been shown to be due to a reduced output of this hormone, for when it is administered to young dwarfs they grow to normal size [18]. Growth hormone has also been shown to increase nitrogen retention by the body and so to increase the efficiency of conversion of protein in the feed to body protein. Unfortunately its cost of production from ox pituitaries is very high, and no one so far has been able to find a cheap substitute, as has been done for some of the other anterior pituitary hormones; the follicle-stimulating hormone, for example, can be replaced by a substance found in the blood serum of the pregnant mare. Nor has it yet been possible to synthesize the hormone, so that its direct application to commercial meat production is not practicable. Nevertheless, it is highly probable that the output of growth hormone in the animal can be increased by implanting or feeding synthetic oestrogens.

EFFECT OF OESTROGEN TREATMENT ON LIVEWEIGHT GAIN

Following work on the treatment of poultry with synthetic oestrogens, which showed that breast muscle fat may be doubled and juiciness of the meat improved by injection of oestrogens into the neck, experiments were started in the U.S.A. on cattle and sheep. In 1954 the U.S. Food and Drug Administration permitted the use of 10 mg. of stilboestrol daily in feeds for fattening cattle weighing 600 lb. or more, and from then onwards it came into general use. Byerly [5] estimated that in the U.S.A. in 1955 some 5 million cattle were fed on rations that contained oestrogen.

Numerous experiments have been made in the U.S.A., and a few examples may be quoted. Burroughs, Culbertson and Kastelic [4] reported that in experiments at nine agricultural experimental stations in which stilboestrol was fed to 293 cattle and 255 served as controls, the average daily gain of the controls was 1·97 lb. and that of the treated animals 2·34 lb. Klosterman and others [13] implanted stilboestrol into steers and bulls and obtained a daily gain of 2·79 lb. as compared with 2·08 lb. made by the controls. Mitchell and Armstrong [15] fed steers equal amounts of concentrates and found that the controls grew at the rate of 2 lb. per day and took 124 days to reach killing weight, whereas the treated animals gained 2·5 lb. per day and took only 96 days. Moreover, the stilboestrol-fed steers required only 665 lb. of concentrates per 100 lb. gain, whereas the controls required 846 lb. Thus rate of gain and efficiency of food conversion were improved.

Extensive experiments have also been made in Great Britain during the last few years, mainly at the Rowett Research Institute under the direction of Mr. J. A. Crichton and at the School of Agriculture, University of Nottingham, under the direction of Dr. G. E. Lamming. This work has in general confirmed the U.S.A. results. It has also given some indication of the mode of action of the hormone and the effects on carcass composition, and has provided information on the circumstances under which it can best be used and pitfalls that may arise in its application.

Of the two synthetic hormones in common use hexoestrol is probably better than stilboestrol, for it has less harmful side-effects if too much is given, and a very much larger dose of it is required to have any effects on human beings. Tests made on the flesh from stilboestrol-treated cattle [17] and pigs [3] have shown that no residue of the hormone is present in the tissues and that the meat is perfectly safe for human consumption.

Two methods of administration may be used. The hormone can either be injected in pellet form into the base of the ear, a part which is cut off from the edible flesh when the carcass is dressed at slaughter, or it can be fed in the concentrated ration. When cattle and sheep are at grass in summer injection only is possible, but during winter the feeding method may also be used. Implantation is done as in poultry with an injection needle, and some form of crush is necessary to hold the animal during the operation. In pigs, because the ear is edible, the hormone should be fed. In feeding it should preferably be used in pellets or cubes, as in meals there is danger of uneven mixing

TABLE I

EFFECT OF HORMONE IMPLANTATION ON FATTENING CATTLE
(Data from Lamming [14])

Animals (No.)	Feeding	Dose * (mg.)	Av. daily gain (lb.)		Increase over controls (%)
			Treated	Controls	
17 steers	Winter yard	60	2·00	1·21	63
23 steers	Spring pasture	36	2·73	2·42	15
62 steers	Summer pasture	24	1·60	1·24	33
		36	1·90		53
		30 *x*	1·70		46
20 steers	Autumn pasture	36	2·60	1·70	55
21 heifers	Autumn pasture	36	2·10	1·80	15

* All stilboestrol except *x*, which is hexoestrol.

and of contaminating other feeds for stock for which the hormone is not suitable.

Some effects of implanting hormones into cattle in Great Britain are shown in Table I from some of Lamming's earlier experiments [14]. So far as the experiments have gone in this country, the best dose of hexoestrol to feed to cattle appears to be 10 mg. per day; this amount can be incorporated into 3 lb. of nuts. When implanted, 60 mg. hexoestrol is required for yarded steers, and, for cattle at pasture, 45 mg. for steers and 35 mg. for heifers. It has been suggested that the smaller dose necessary to obtain maximum results on pasture, and especially on spring pasture, is due to the fact that pasture contains some oestrogens. The estimated cost of implantation is about 2s. per head.

The best time to implant is about 100 days before the cattle are expected to be finished; administration to store cattle or young stock appears to be of doubtful advantage. This also applies to sheep, for which the recommended dose for implantation is 15 mg. and the cost about 4½d. per head. Although the rate of liveweight gain in suckling lambs may be speeded up slightly, this is more than offset by a loss in carcass quality [2, 9]; the reasons for this will be discussed below. Some of the earlier results obtained by Lamming [14] in sheep are shown in Table II.

Correct dosage is very important. In many of the earlier experiments too much oestrogen was used, and this not only failed to improve growth beyond that caused by low doses but had harmful direct effects on special parts of the body. This was so particularly where stilboestrol was used. It is well known that large doses of stilboestrol will stimulate udder tissue to develop in virgin heifers and bring them into milk [10] and also cause growth of the nipples in steers and wethers. Excess of oestrogens in the body, as in cows with ovarian cysts and cows about to calve, relaxes the pelvic ligaments and raises the tail-head; such symptoms have been observed in steers implanted with excessive doses of stilboestrol [17]. Inordinate doses may also cause extrusion of the rectum in both sexes, prolapse of the vagina in females and enlargement of the secondary sexual glands in males, making urination difficult or impossible [2]. It is therefore important to use only the minimal doses required to produce the full indirect growth effects without bringing into play the direct effects of the hormones on other tissues.

TABLE II

EFFECTS OF STILBOESTROL IMPLANTATION ON HOGGETS
(Data from Lamming [14])

Animals (No.)	Feeding	Duration of treatment (days)	Dose (mg.)	Av. daily gain (lb.)		Increase over controls (%)
				Treated	Controls	
12	Rape	90	12	0·540	0·453	19·2
34	Swedes	30	12	0·278	0·209	33·0
60	Kale + Concentrates	90	12	0·425	0·349	21·7
112	Turnips + Concentrates	150	12	0·108	0·076	42·1

EFFECT OF OESTROGEN TREATMENT ON CARCASS QUALITY

From the butcher's point of view it is important to know the composition of the increased liveweight, and several experiments with practical and scientific objectives have been made on this point, though there is still much more to be done. In one practical test on high-quality Aberdeen Angus cattle [12] the proportion of muscle to fat was considerably higher in the hexoestrol-treated animals than in the controls (Figure 2). If further research shows that such results can be consistently attained this hormone should have wide applications in practice, for trends in consumer demand are moving rapidly towards more lean and less fat in meat. Extensive experiments on cattle and sheep [7, 14] have shown that there is a slight decrease in fat but that the gain in weight consists largely of lean meat and bone with some moisture.

The fact that hexoestrol stimulates the growth of muscle and bone and not that of fat suggests where the treatment is likely to be of value in practice. The consumer now wants leaner meat than formerly, so the treatment of older steers which tend to put on too much fat is indicated. Similarly heifers and, in particular, old cows which often have too much fat in proportion to lean are marked out as most suitable animals for treatment. Good results have been reported with old cows [11] and old ewes [7].

As the meat of old animals tends to eat dry unless impregnated with fat, it seems likely that the extra water in treated carcasses, located probably in the connective tissues as well as in the muscles, will improve the eating qualities of meat from old animals. On the other hand, the treatment of young growing animals, such as fat lambs, where the main difficulty in practice is to get enough fat on the animal to meet consumer demand, will lower carcass quality and more than counterbalance the small extra gain in liveweight. For store stock it would also appear to be unsuitable, for they tend to grow more bone than anything else and implantation would only add to this.

Little work on hexoestrol feeding has yet been published on pigs, although some is in progress. Hog pigs have more fat and less lean than gilts of the same age and weight, and it would therefore be useful to know whether hexoestrol feeding would improve their carcass quality.

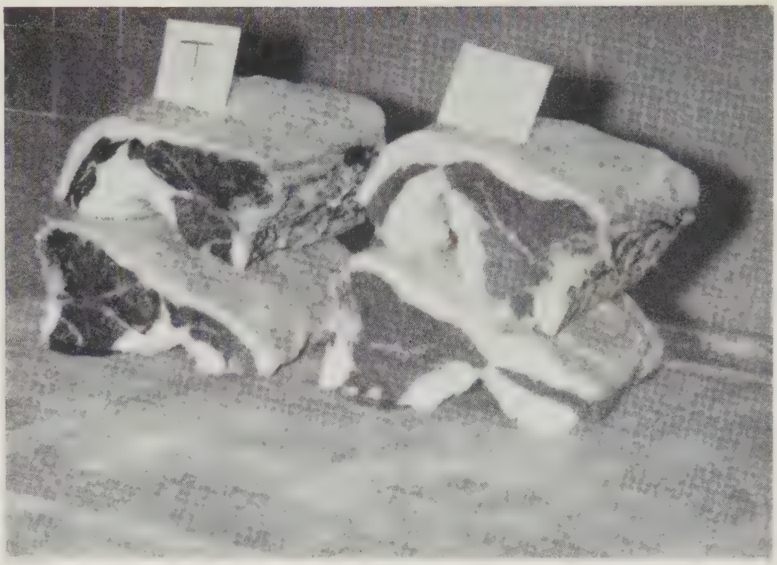

FIG. 2. Sirloins from Aberdeen Angus steers. Left: Treated with hexoestrol. Right: Control. The additional proportion of lean meat which is well marbled is seen in the treated animal, while the quantity of suet is greater in the control. (From Justice [12].)

MODE OF ACTION OF OESTROGENS

How the low doses of oestrogens act to produce the increase in liveweight and differential growth rate on the different tissues of the body is not yet fully understood. It is evident that the effect is not a direct one, for heifers which produce oestrogens naturally are usually smaller and fatter than steers which produce no oestrogens. Moreover, if the effect were direct it should increase with the dose given, yet this is not so with increased liveweight gain. It is only when the dose is raised that the direct effects of the oestrogens—growth of udder and nipples and raising of the tail-head—are observed. At present there are two theories concerning the mode of action.

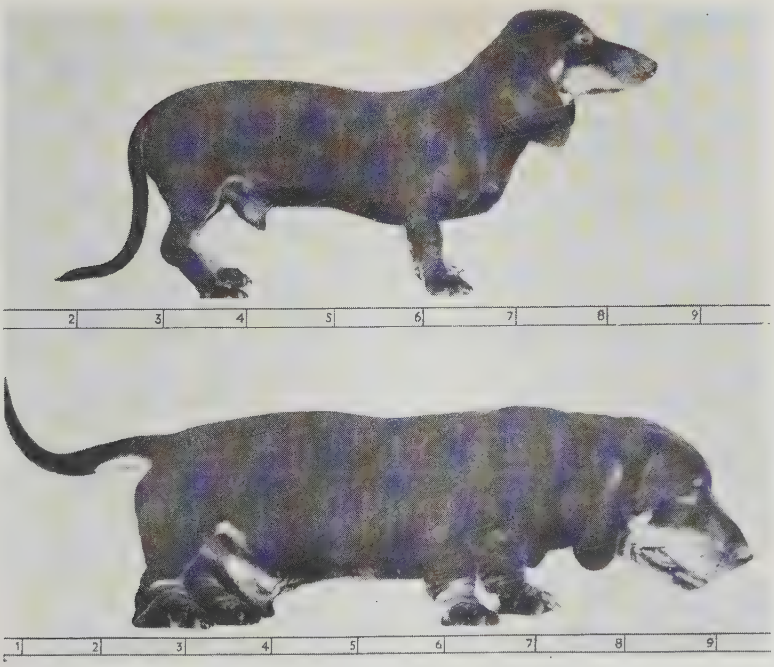

FIG. 1. Changes in growth produced in the dachshund by the administration of anterior pituitary growth hormone. The animals shown are litter brothers. Above: Normal, untreated. Below: Treated. (From Evans and others [8].)

The first theory, put forward by Clegg and Cole [6], is that oestrogens act on the adrenal gland and cause it to produce the male hormone, testosterone, which is responsible for the greater muscular development of the bull as compared with the steer. To test this supposition some investigators implanted steers with testosterone and found that it did increase the rate of gain in liveweight, but the rate was lower than with hexoestrol and not higher, as might have been expected had the oestrogen worked in this way. If the implanted oestrogens acted by producing testosterone, then the muscles of the neck would presumably have increased more than those in other parts of the body; but the few tests that have been made provide little or no evidence of this—in fact the increase appears to be at least as great in the muscles of the loin as in those of the neck. Bull meat has more of the red colouring matter, myoglobin, in the muscle than meat from steers, and this suggests that if oestrogens acted through the production of testosterone the muscles would be darker in colour in treated animals than in untreated animals. In some experiments done by the U.S. Agricultural Research Service, however, no difference was observed in the colour of the meat from treated and control groups. Available evidence therefore seems to be against this theory.

The second theory is that the oestrogens stimulate the anterior pituitary gland to secrete more growth hormone, possibly indirectly by damping down the production of gonadotrophic hormones. It is well known that growth hormone increases the retention of protein in the body and has no effect on the conversion of energy into fat. Growth hormone also increases the growth of bone. Other supporting evidence for the theory is that administration of growth hormone has a marked effect on the growth of the skin (Figure 1). The skin of young, quickly growing animals is particularly supple, owing to the amount of water in the connective tissues. Several observers have claimed that they could pick out by eye treated from control animals at pasture. Byerly [5] noted that the pelts of treated sheep were a little heavier, and Lamming found the same thing.

On the evidence now available it would appear that administered oestrogen acts by increasing the output of growth hormone and thereby makes the animal temporarily younger, when it normally grows more bone and muscle and less fat and its tissues contain rather more water. If this is so, an improvement in meat quality might also be expected, for when the animal is young the connective tissue binding the muscle cells together has a high water content and so is comparatively tender, whereas in the ageing animal the connective tissue loses water and becomes tough. Whether oestrogen treatment will rejuvenate the meat of old animals has yet to be put to scientific tests, but the present evidence indicates that it may do so.

ACKNOWLEDGMENT

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RESISTANCE TO INSECTICIDES

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Many problems that are difficult to solve have been created by the appearance and spread of insecticide-resistant strains of insects. We now know that resistance is inherited, rapidly acquired and slow to be lost, and ways have been indicated in which it may arise; but more must be discovered about the mechanism of action of the toxicants and their metabolism in the insect body before the nature of the trouble can be fully understood.

A hundred years have elapsed since Darwin in *The Origin of Species* presented evidence that inherited differences in living things came about by a process of natural selection of more or less random variations. Such evolution, however, takes place very slowly, and it is rare for its results to become manifest in the course of a few years. From this point of view, the widespread appearance of insecticide-resistant strains of insects shortly after the introduction of powerful synthetic insecticides is of great scientific interest. It also creates an important commercial problem. Literature on the subject is extensive and has been well reviewed [1, 2, 12, 20].

Insecticide resistance is not strictly a new phenomenon. As long ago as 1897 Smith [24] noticed variations in the effectiveness of kerosene against the San José scale insect (*Aspidiotus perniciosus*) in different areas, but it was not until 1914 that insecticide-resistant insects began to attract attention. In that year Melander [19] found that San José scale could no longer be controlled by lime sulphur, though oil sprays remained effective. This report was received with scepticism, yet eleven years later lime sulphur had to be applied at ten times the original concentration to give satisfactory control of the pest, and other instances of the development of resistance began to be brought to light. To cite only two of these, Quayle [22] in 1916 reported that the California red scale (*Aonidiella aurantii* Mask) and the black scale (*Saissetia oleae* Bern.) had become so resistant to hydrogen cyanide that fumigation had to be adopted annually instead of once in every three years, and Du Toit [10] in 1938 found the cattle tick (*Boophilus dicoloratus* Kch.) to be resistant to arsenic—a dip that killed 99% of a normally susceptible strain destroyed only 37% of the resistant population.

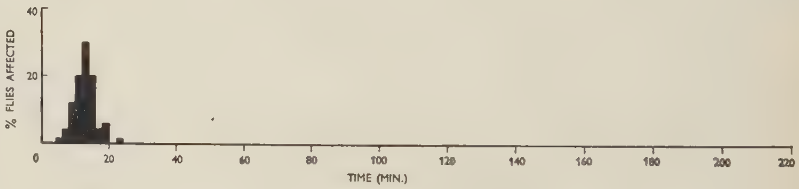
These examples show that entomologists were familiar with insecticide resistance, though not as a major problem, long before the introduction of the persistent contact insecticides DDT and BHC set an altogether higher standard in the chemical control of insects. In the field of public health, for the control of insect-borne diseases, the prospects of the new materials seemed at first to be particularly bright. A single application leaving a minute deposit on the inner surface of a building provided good mosquito and fly control for several months, and relatively cheap and effective prevention of malaria and of fly-borne diseases was practical. Yet disquieting reports were soon to appear.

THE DEVELOPMENT OF RESISTANCE

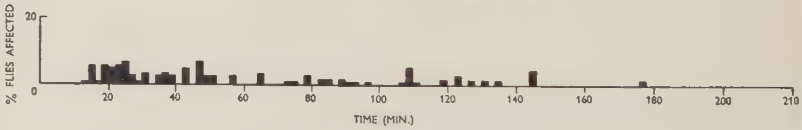
The failure of DDT to control houseflies was first reported in Italy by Sacca [23] and by Missiroli [21] in 1947. It is not, perhaps, surprising that the discovery should have come from that country, for it was there that the first widespread use of DDT was made by the American Occupation Force against malaria-carrying mosquitoes. After an initial use in 1944, DDT residual sprays were extensively applied in 1946 and in the following year the first DDT-resistant houseflies appeared. At about the same time, reports of resistance to DDT came from Sweden and from Denmark. Weismann collected flies at Arras in Sweden and found them to be about 100 times as resistant to DDT as normal flies. In Denmark [13] DDT had been widely used on farms, and by the summer of 1947 fly control had failed in many parts of the country. Probably the best-known cases of resistance, however, are those which occurred in Greece [11] and Sardinia [3] following the well-organised campaigns of malaria control by mosquito eradication that had been put in hand shortly after the end of the war, using DDT residual deposits as the main treatment against adult mosquitoes: initially, excellent housefly control was obtained as an effect incidental to the mosquito control, but about two years later flies reappeared in large numbers and were almost everywhere highly resistant to DDT.

Where DDT failed attention was naturally directed to alternative insecticides. In Denmark and Sardinia, DDT-resistant flies were well controlled at first by BHC and chlordane, but after an interval of 1–2 years resistance to these compounds again made fly control difficult. In Sardinia by 1950 hordes of flies were present in the villages, and tests carried out later that year showed that they were resistant not only to DDT, BHC, chlordane and toxaphene, but to aldrin and dieldrin, which had not been used in the field.

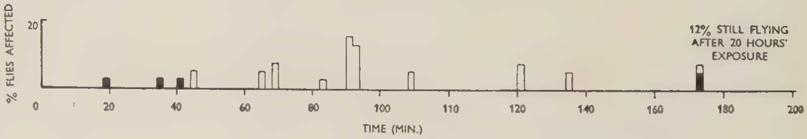
Before discussing the subject further, some consideration must be given to the measurement of resistance. It is usual to define the potency of an insecticide to a given insect population by its LD50 value (i.e. the dose that will kill 50% of the individuals exposed to it), and such values have often been used to measure changes in resistance. The interpretation of "resistance" figures of this kind is, however, not quite straightforward, for there may be a change in the distribution of resistance within the population. This may be demonstrated by exposing flies continuously to an insecticide and recording the times at which individuals become affected by it. Histogram (*a*) shows the result of such a test on a normal fly population in which the numbers of flies becoming affected at intervals of 2 minutes are plotted against exposure time. Histogram (*b*) shows a similar record for a strain from Sardinia in which the distribution of resistance is markedly different.



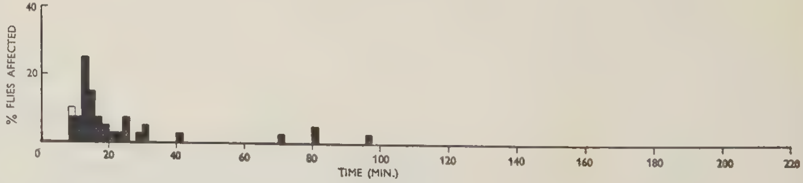
(*a*) *Normal susceptible strain*



(*b*) *Resistant strain from Sardinia*



(*c*) *Resistant strain from Uruguay after laboratory selection*



(*d*) *Resistant strain from Uruguay after seven years without selection*

Effect of BHC on various strains of flies, showing (black columns) flies knocked down and killed, and (white columns) flies knocked down but recovering within 24 hours.

Extensive field and laboratory experiments have now made it clear that, so far as resistance is concerned, the chlorinated hydrocarbon insecticides fall into two groups: one including DDT and closely related compounds such as methoxychlor, and the other such substances as BHC, chlordane, aldrin and dieldrin. Resistance produced by selection with any one compound confers some degree of resistance to other members of the same group, but is independent of resistance to compounds of the other group. Resistance in either group is not related to resistance to completely different compounds, such as the organic phosphates.

LABORATORY SELECTION EXPERIMENTS

In practice, resistance to insecticides of these two groups may become important after only 2–3 years of their intensive use in the field. Presumably the factors responsible are already present in a small proportion of the natural population or arise by a mutation which occurs relatively frequently, and subsequent selection by the insecticide increases the proportion of resistant individuals. In relation to the development of resistance in the field, the results of selection experiments in the laboratory are to some extent conflicting.

Various fly strains have been subjected in each generation to doses of insecticide that leave a few survivors to carry on the race. If a strain that has already acquired some degree of resistance in the field is exposed to selection in this way, a very high resistance is rapidly developed (histogram (*c*)); but if a normally susceptible strain is used, resistance is not always easily produced. American workers [16, 30] report that selection of flies as adults through 14 generations with DDT sprays produced an unusually strong stock, rather than one having a specific resistance to DDT. Bovingdon [4] attempted to breed a strain of flies resistant to gamma-BHC by adding this chemical to the larval culture medium, and carried the line through 38 generations without the insects becoming significantly more resistant than they were originally. In these cases the laboratory populations may have been too small to contain the necessary genetic factors for resistance to develop, and hence no amount of selection could produce a specific resistance. There are, however, numerous records in which laboratory selection has produced specific resistance from an apparently normal strain; but synthetic insecticides, particularly DDT and BHC, have been used so extensively that it is now difficult to obtain a field strain of flies entirely uninfluenced by insecticide selection.

REVERSION BY RESISTANT STRAINS

Of great practical importance is the rate at which a resistant strain will revert to susceptibility in the absence of exposure to insecticides. Where control has failed through resistance, how long will it be before the insecticide can again be successfully used? This point is also of considerable theoretical interest. If a resistant fly differs from a susceptible one solely in its resistance to the particular chemical, then lack of resistance carries no survival value in the absence of insecticide; there is, in fact, no selection pressure towards susceptibility, and reversion should be very slow. The physiological differences upon which resistance depend may not be entirely independent of other physiological and even morphological differences which may permit selection pressure by the normal environment in the direction of susceptibility. At present insufficient is known of other variations associated with resistance to enable any useful forecast on reversion rate to be made.

Field and laboratory studies usually show that the rate of reversion is slow. March and Metcalf [18] found no loss in a DDT-resistant strain reared for 35 generations in the absence of DDT. Knipling [15] collected from the field six colonies of flies that were 20–50 times as resistant to DDT as a susceptible strain, and, after rearing for six generations in the absence of DDT, found that most of them still showed some resistance, though the extent of the loss varied greatly in the different strains. In the author's laboratory at Jealott's Hill Research Station a strain of flies resistant to gamma-BHC, obtained from Uruguay in 1950, has been maintained for seven years (through 140 generations) without contact with BHC, yet the greater proportion of the flies still show some degree of resistance. Histogram (*d*) shows that the distribution of resistance in 1957 is still different from that of a normal strain (histogram (*a*)).

Laboratory tests on reversion, even if carried out over many generations, are of limited value in forecasting what may happen in the field. In a laboratory culture the flies are in a protected environment, and factors possibly disadvantageous in the field may have no effect. Field experience provides little evidence of any significant loss of resistance to DDT.

THE MECHANISM OF RESISTANCE

Resistance to an insecticide could possibly come about in a variety of ways. For instance, the amount of insecticide taken up by the insect might be decreased because an alteration in the structure or composition of the cuticle reduced the rate of penetration. Weismann [28] noticed that in DDT-resistant flies from Sweden the pulvilli on the feet were stouter and the articular membranes of the tarsi were thicker than on susceptible Swiss flies, and he suggested that resistance might be due to the more difficult passage of DDT through the thicker cuticle. March and Metcalf [17], however, have shown that, when DDT solution was injected directly into the body cavity of resistant and normal flies, the relative resistances were the same, and hence the effect of the cuticle was unimportant. The differences noted by Weismann were, in fact, differences between Swedish and Swiss strains of flies not necessarily related to resistance.

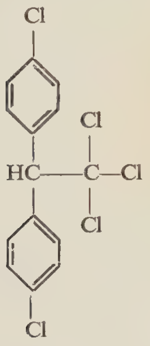
Uptake of insecticides might also be influenced by altered behaviour. King and Gahan [14] observed that resistant flies tended to settle on untreated feed troughs and dairy floors in preference to treated walls and ceilings. Busvine [9] considered that resistant flies might be irritated sooner than susceptible ones and be knocked down more readily, so preventing the accumulation of a lethal dose. Behaviour differences in flies seem to exist and may play some part in resistance, but injection experiments show that uptake is not a major factor.

If the insecticide is present in the insect, resistance could be due to a difference at the site of action, i.e. a difference in the biochemical or physical system with which, in the susceptible insect, the insecticide interferes. Here the difficulty is that we know very little about the ultimate chemistry and physics of the action of the BHC and

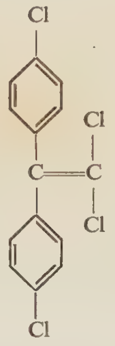
DDT groups, resistance to which is of greatest importance.

Resistance could also develop if the insecticide, although entering the insect, is prevented from reaching the site of action, either by being rapidly converted to non-toxic substances or by being stored in places where it does no harm. The first of these possibilities is the easier to investigate, and much work has been done on the metabolism of both DDT and BHC in resistant flies.

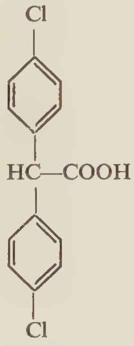
Living things in general are equipped with chemical mechanisms for dealing with a great variety of organic chemicals. White and Sweeney [29] administered DDT to rabbits and showed that it was converted first to the non-toxic compound DDE and then to the water-soluble DDA, which was eventually excreted in the urine. The reaction is indicated in the diagram below.



DDT
2:2-bis-(*p*-chlorophenyl)-1:1:1-trichlorethane



DDE
2:2-bis-(4-chlorophenyl)-1:1-dichloroethylene

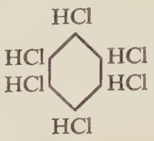


DDA
bis-(*p*-chlorophenyl) acetic acid

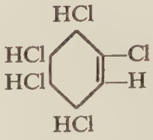
The mechanism in flies has been investigated in greater detail by Sternburg and Kearns [25, 26]. They found that resistance is due to the ability of the strains to convert absorbed DDT to the non-toxic DDE, though the further conversion to DDA does not occur. They also extracted the responsible enzyme from resistant flies and demonstrated its ability to effect the conversion apart from the fly; in these circumstances glutathione is an essential cofactor. The same enzyme is able to detoxify methoxychlor, though less readily than DDT.

The conversion of DDT to DDE may not, however, be the complete explanation of resistance in flies, for Winteringham [31] has shown that certain DDT-resistant strains were also resistant to Prolan (1 : 1-*bis*-(*p*-chlorophenyl)-2-nitropropane), which cannot be converted by dehydrochlorination to DDE.

The mechanism of BHC resistance has been investigated by Bradbury [6–8] and by Sternburg [26]. Sternburg has described experiments which indicate that resistant flies may dehydrochlorinate gamma-BHC to give pentachlorocyclohexene:



BHC
benzene hexachloride



pentachlorocyclohexene

Using radiochemical methods, Bradbury and Standen compared the metabolism of BHC in susceptible and resistant flies. In both strains a portion of the absorbed BHC was converted into water-soluble products, but the rate of conversion was greater in the resistant strain. Resistant flies also absorbed less BHC than susceptible ones, and the combined effect of reduced pick-up and increased decomposition was to reduce the BHC in resistant flies to about a quarter of that in susceptible ones. More recent work by the same authors [5], using ^{36}Cl- and ^{14}C-labelled BHC isomers, showed that metabolism produced chloride corresponding to the removal of 4–6 chlorine atoms for each molecule of BHC, and 11 different water-soluble compounds. They were unable, however, to demonstrate the existence of pentachlorocyclohexene as an intermediate stage.

Bradbury and Standen also measured the capacity of a number of BHC-susceptible species, including beetles, locusts, mosquitoes and houseflies, to metabolise BHC isomers. The susceptible housefly shows exceptional ability to eliminate gamma-BHC as water-soluble products, and it is possible that this may be related to the widespread ability of flies quickly to become resistant to poisoning by gamma-BHC. The existence in a normal fly of a metabolic pathway for dealing with BHC may predispose this insect to the acquisition of insecticide resistance.

THE PROBLEM IN GENERAL

Undoubted instances of resistance have been recorded in a number of insect types other than houseflies. Among the important disease-carrying mosquitoes the development has fortunately not been rapid, but certain strains of *Anopheles gambiae*, which is the most important malaria vector in Africa, have recently become resistant to dieldrin and hence to BHC and other materials of that group. Resistance to DDT and BHC, as well as to arsenic, is important in the cattle tick, *Boophilus*, particularly in South Africa and Australia. Lice resistant to DDT occurred during the Korean war. Among plant pests, red spider mites have become highly resistant to organic phosphorus insecticides in many areas.

It is possible that the action of a chemical against a resistant insect may be aided by a synergist, i.e. a chemical that can increase the effect produced by a toxicant without necessarily being toxic itself. Synergists may be chemically related to the substances whose action they aid, and some are believed to act by combining with the enzyme systems that normally detoxify the active material. To be effective, however, synergists would have to be applied in quantities at least comparable with those of the active material, and would have to possess similar physical properties to enable them to penetrate in adequate amount to the sites of the detoxifying enzymes.

The widespread occurrence of insecticide-resistant insects undoubtedly presents a formidable problem, and even a threat to our existing methods of insect control. It seems likely that as more time for selection passes more and more pest species will become resistant. Control of insects by small amounts of highly active chemical is convenient and cheap in comparison with cultural and hygienic precautions in agriculture and public health, but it is now appreciated that insecticides must be used intelligently in addition to, and not to the exclusion of, other methods. The development of more selective insecticides seems desirable, particularly of materials that do not unduly interfere with the role of predators in the natural control of pest populations on certain crops. Predator action could be important in delaying or preventing the onset of resistance by removing the few resistant individuals that remain after an insecticide application.

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THE BREAKDOWN OF IMMUNITY FROM POTATO WART DISEASE

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It has always been assumed that the wart disease fungus exists everywhere as a single homogeneous race to which potato varieties are either susceptible or immune, and government lists of recommended immune varieties have been accepted internationally. Recently new aggressive races have appeared which can attack many if not most of these immunes. This is a threat that could become serious and the position is being closely watched.

The fungus that causes wart disease of potato, *Synchytrium endobioticum* (Schilb.) Perc., was first described by Schilberszky [14] from Trentschen, now part of Czechoslovakia, but later observations showed that the disease must already have existed for some time both there and in other countries, including Britain. Whatever its origin, the disease soon became a real menace in many parts of Europe and something to be guarded against in other parts of the world. Most countries in which the disease has gained a footing have introduced domestic legislation to check or prevent its further spread, and there is now scarcely a country which does not include special requirements about wart disease in its regulations governing plant imports. When the European and Mediterranean Plant Protection Organization (E.P.P.O.) was established in 1950, wart disease was one of the five scheduled pests and diseases—and the only disease—to which, under the Convention [2], it was agreed the Organization should pay particular attention, and in the following year E.P.P.O. issued recommendations [1] about the general principles on which import restrictions concerning the disease should in future be based.

DISTRIBUTION AND ECONOMIC IMPORTANCE

Before the 1939–45 war potato wart disease had, at one period or another, become widely distributed and locally destructive in central Europe, Scandinavia, the British Isles, and to a less extent in Holland and southern Finland. Localized attacks had occurred also in Belgium and France, but the Mediterranean area remained free. Outside Europe the disease was confined to a few small areas in South Africa, the central Andes in South America, certain mining districts of Pennsylvania and adjacent States, and the island of Newfoundland, where it was already severe. Isolated outbreaks had occurred in Canada, Portugal and Algeria but had been eradicated.

Since 1940 the disease has been recognised in a few of the European countries bordering on the old infected central region, including Lithuania, Russia, Rumania, Yugoslavia and northern Italy, but the Mediterranean area still remains free. There appears to have been little or no extension outside Europe. In South America it has been seen also in Uruguay, and now occurs in the Falkland Isles. It has still not been found in Australasia or Asia, except for a single record in West Bengal in 1953, when it was introduced with imported seed potatoes.

During the first thirty years of this century wart disease caused alarm and heavy loss in those countries where it became firmly established. On the whole it was most destructive in mountainous regions of high rainfall, around large cities, and in industrial areas where there are usually many smallholdings on which potatoes are grown year after year for local domestic consumption.

The situation was ultimately saved as a result of the combined efforts of plant breeders and legislators, following the discovery in 1909 by Gough [7] that certain varieties, notably Snowdrop, remained unaffected even in badly contaminated land. In Britain, as elsewhere, plant breeders concentrated on the production of new varieties immune from the disease, and as these became available it was possible to add to existing legislative measures, designed to check further spread, one which enforced the growing of immune varieties on infected land. Altogether there have been about 13,500 recorded outbreaks of wart disease in England and Wales alone, of which over 10,000 were reported before the Wart Disease of Potatoes Order of 1923 came into effect. The vast majority of these have occurred in gardens and allotments, and, though no county has remained free from the disease, in Britain as a whole the black spots have been in the west midlands, north-west England, and mid- and south Scotland.

Under the 1923 Order the only potatoes that could be planted in land on which wart disease had been known to exist at any time were approved immune varieties, and this had an immediate effect on the spread of the disease. Before 1923 hundreds or even thousands of new outbreaks were reported each year; but since 1929 the number has rarely exceeded 100 and then only after exceptionally wet summers, such as those of 1931 and 1936. The cultivation of immune varieties gradually became common practice, irrespective of legislation, and now that some two-thirds of the potatoes planted in this country consist of immune varieties (King Edward is the only important exception) the losses from wart disease are slight and the disease is of little economic significance. This is true also of other countries where the disease has been widespread. The importance of the disease now lies, or did lie, more in the fear of what it might do in countries hitherto free or practically free from it than in the crop losses it actually causes.

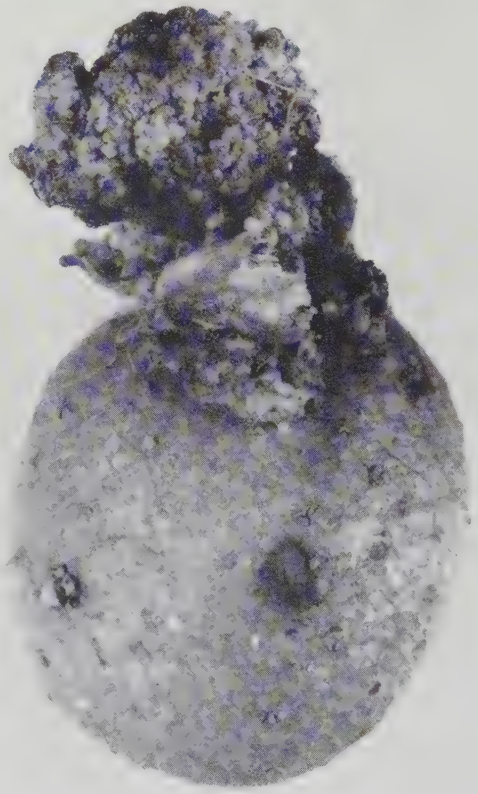
APPEARANCE OF NEW PHYSIOLOGIC RACES

The complacent attitude towards wart disease fostered by the general adoption of immune varieties has now been rather rudely shaken by the discovery of new physiologic races of *S. endobioticum* which are much more aggressive than the old one and which, indeed, seem to be capable of attacking most of the varieties now listed as immune. Wart disease was long regarded as the only fungus disease against which there was absolute varietal immunity, as distinct from high varietal resistance. Some doubt was thrown on this unique distinction when laboratory testing for immunity was devised, because it was found that certain varieties hitherto listed from field trials as immune became very slightly infected when given the more delicate indoor test. However, only the superficial tissues are affected and these are soon sloughed off, so that the varieties are in fact field immune. Apart from very occasional differences of opinion about the susceptibility or immunity of a particular variety there has been nothing to suggest that more than one race of the pathogen existed or that this race behaved differently in different countries.

This has not been the experience with other fungus diseases. The potato varieties so far bred for resistance to blight (*Phytophthora infestans* (Mont.) de Bary) have invariably proved partially susceptible within a year or two because the fungus exists in a number of differing races or mutant forms, and when a new potato variety is resistant to the common races others less common will multiply upon it. Varieties of tomato bred for resistance to leaf mould (*Cladosporium fulvum* Cooke) have gone the same way, and the strawberry variety Auchincruive Climax, which for a time was highly resistant to the soil-borne red core disease (*Phytophthora fragariae* Hickman), is susceptible to new physiologic races. Now the same thing is happening with the wart fungus, and although the true position is still rather obscure the following sequence of events is sufficient to indicate that the matter cannot be lightly dismissed. The information given is based solely on published literature and is liable to be incomplete.

Germany

The first hint of fresh trouble came when new races of *S. endobioticum* were reported from Central



A potato tuber of the variety Arran Chief affected with the normal type of wart disease.

Europe. Braun [5] related how a field of the immune variety Ostbote became totally infected with wart disease at Giessübel in Thuringia in August 1941. In his artificial infection tests Braun could still not infect this variety with the ordinary race (the Dahlem or D-race) used in the official German immunity tests, but it readily became attacked by the new race present in the affected field, which he called the G-race from the place where it was found. Moreover, in further trials only two (Fram and Frühe Hörnchen) out of 67 immune varieties grown commercially in Germany proved resistant to the G-race, which also attacked 37 out of 40 other immune varieties not grown commercially. By the summer of 1943 an appreciable area around Giessübel was infected, and three years later traces of the new race were detected in the adjacent district of Oberneubrunn. Meanwhile, in 1942, further outbreaks of this or another aggressive race had occurred on the previously immune varieties Ackersegen, Flava and Ostbote in fields at Pappenheim, 25 miles distant from Giessübel [8].

The problem was tackled promptly. The new race was included forthwith in the official wart immunity trials for seedlings, stringent measures were taken to prevent the further spread of the G-race, and it was made obligatory to plant the variety Fram—a rather poor yielder—in the contaminated land as a temporary measure. In view of these measures it was thought that the likelihood of even a local major disaster was remote [15]. In 1949 a plan of campaign was drawn up to deal with the two infected areas. A quarantine ring with a radius of 5 km. was to be defined in which varieties and breeding lines immune from the new races were to be planted pending the availability of varieties that would combine immunity with maximum productivity. All new outbreaks of the disease would be carefully tested for the presence of the new aggressive races (for these had appeared in areas where the old one was widespread), and by 1960 there was to be an embargo on susceptible varieties in the affected district.

Unfortunately a fresh centre of infection was found in Thuringia at Rudolstadt in 1950 and the following year the new race appeared at Koppatz, about 150 miles further to the north-east [10]. Even more discouraging was the discovery of aggressive races at a number of widely separated districts in western Germany. The first of these outbreaks was detected in 1950 on the varieties Ackersegen and Bona at Dorsten in Westphalia. The second occurred a year later at Mengede, about 20 miles to the south-east [16], and in 1952 further outbreaks were confirmed at Olpe, Siegen and Altena, situated at varying distances up to about 70 miles to the south-east of Dorsten [17].

There appears to be no published information about further outbreaks in Germany since then,* and it is still not known how many new races are involved. It is, however, clear that aggressive races which can attack most of the hitherto immune varieties are widely distributed in Germany. Field trials have shown that very few of the existing varieties are immune from these races, and although 100 or more of the thousands of breeding lines tested have proved to be immune, many of these are of little agricultural value [10, 17]. One surprising result has been that some varieties which are immune from the new races in laboratory tests have proved susceptible in the field [9]. This is contrary to experience with the old race, but may be merely a result of imperfect laboratory techniques when using the new races.

Czechoslovakia

About the time Braun made his discovery in Germany, Blattný [3] reported on a study he had made of a considerable amount of warted material collected in Central Europe. He too found new and aggressive physiologic races, and he differentiated them into three groups. One of these he called the SB or mountain race group, which he found in collections from widely separated mountainous regions of Czechoslovakia, including southern Bohemia, eastern Moravia, the Carpathians, and perhaps also from Vorarlberg in western Austria. He thought this SB-race might be the same as the G-race, but Braun [5], to whom Blattný sent material, concluded that the two were distinct and could be distinguished on the basis of their different behaviour on a number of test varieties. Blattný's second group was the east German lowlands race, which he found in northern Bohemia; and his third one, the west German race group, was possibly identical with the first. This appears to be the only published data about the occurrence of new aggressive races in Czechoslovakia, and their importance is not known.

Italy

Wart disease was not reported at all in Italy until 1941 [4], when it was found in the Chiavenna mountain region near the Italian–Swiss frontier, where it may have been present for some years. A special survey in September 1947 revealed that the disease was widespread in most potato-growing areas around Chiavenna at altitudes over 800 m., and the fact that the hitherto immune variety Ackersegen was severely affected [12] strongly suggests that an aggressive race was present. Since then small outbreaks have been found about 100 miles away at Trento (1952) and Bolzano (1955).

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* But see *Z. PflKrankh.*, **64**, 1957, 452–7, and *F.A.O. Plant Prot. Bull.*, 5, 1957, 181–7.

Russia

Here again wart disease has been seen only in the last decade. It is said still to be confined largely to gardens and smallholdings, but the presence of aggressive races is clear from varietal susceptibility to material collected from Minsk, Cernovicy and Leningrad [13].

Newfoundland

Only two outbreaks of wart disease have occurred in Canada and those were successfully dealt with; both were in gardens, one near Ottawa in 1912 and the other at Halifax in 1941. In Newfoundland, however, where the disease was first confirmed in 1909, no steps were taken to eradicate it, and by 1928 the disease was present everywhere on the island. Rotation of crops is almost impossible on the small plots available, and in the early 1930s an attempt was made to counteract the disease by importing seed of immune varieties. This may have succeeded for a time, but it has become apparent [11] that extremely virulent races must now exist there, for varieties listed as immune in Britain, including Arran Victory, Arran Banner, Arran Pilot, Great Scot and Kerr's Pink, all become heavily attacked the first season they are planted in Newfoundland from Scotland. North American resistant varieties, such as Keswick, Canso and Katahdin, have also broken down in the island, but Sebago has retained it resistance for some years [6].

CONCLUSIONS

New aggressive races of the wart pathogen have so far been recognized in five countries. In Germany they have come to light in restricted areas where potatoes, and especially susceptible ones, have been grown frequently and where the normal form of wart disease has been established for many years. The same is true for the island of Newfoundland. It is under exactly these conditions, when the fungus multiplies rapidly, that by hybridization and selection new races may be expected most readily to arise. But they will not usually be detected until varieties immune from the normal race are grown in contaminated land, so that the distribution of the new races may indeed be wider, and the period during which they have been in existence longer, than the records suggest. In any event, experience in these two areas can be put to advantage, if only by stressing the folly of planting susceptible varieties in land contaminated with the wart fungus, whether the race is an old or a new one.

A different explanation would seem to lie behind the origin of aggressive races in Russia and northern Italy where the disease has not been known previously to exist, but until all the facts are known it would be idle to speculate on this.

It is said to be encouraging that a few commercial varieties and a number of breeding lines appear to be immune from all known races of the pathogen, but this is cold comfort to those countries where new aggressive races may appear at any time, for it merely means putting the clock back forty years and starting breeding all over again.

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THE NATURE OF PLANT VIRUSES

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Current knowledge of the constitution and behaviour of viruses shows that they differ fundamentally from other types of pathogen. They more nearly resemble the components of cells that are responsible for inherited characters than they resemble bacteria, and their formation seems to be a part of the nucleoprotein synthesis of infected cells. This adds to the difficulty of finding therapeutic substances that do not harm the host.

Hackneyed though it is, there seems no better way of introducing my subject than to dip back into the past, to the time when viruses were first discovered, for this historical excursion will show simply and vividly the kind of questions that were almost immediately posed by these elusive entities.

During the second half of last century, mainly because of Pasteur, the germ theory of disease became firmly established and the earlier belief in the spontaneous generation of one kind of organism from another was dispelled. There followed a period during which it was confidently thought that, for each infectious disease, there would be isolated a specific bacterium, fungus or some other micro-organism which could be characterized by its appearance under the microscope or by its growth on culture media. Pasteur's failure to find any such cause for rabies did not greatly shake his confidence that one existed, though it did make him suggest that it might be "infinitely small." Similarly, Ivanowski, who is traditionally credited with the discovery of viruses because he found in 1892 that juice from a tobacco plant with mosaic could still cause the disease after it had passed through a bacteria-proof filter, was so imbued with the idea that an organism must be responsible that he thought it likely his filter had leaked and allowed bacteria through or that the disease was caused by some toxin present in the filtrate and made by the postulated bacterium.

Here, as happens so often in science, though Ivanowski did not appreciate it, more was to be learned from the failure of an accepted technique than had it succeeded. Beijerinck, who soon repeated the filtration experiments, did appreciate it, and he would have none of Ivanowski's explanations. Beijerinck was impressed not only by the filterability and stability of tobacco mosaic virus but also by its ability to diffuse through agar, and he concluded that he was dealing with something quite different from a bacterium—so different that he went so far as to suggest the virus was not "corpuscular" and tried to convey his ideas about it by calling it "an infectious living fluid." Thus our subject was already set in the nineteenth century: not Beijerinck's meaningless problem about whether the virus was a particle, though for some years this was debated seriously by a few people, who were apparently willing to abandon the atomic and molecular theory of the structure of matter, but simply: Are viruses essentially similar to microscopic organisms? Or do they constitute a biological category of their own, fundamentally different from even the smallest bacterium?

As more and more diseases were found that resemble tobacco mosaic in having filterable, invisible causes which fail to multiply on the kinds of media that support most bacteria, the economic importance of viruses became increasingly obvious, even though their nature remained wholly obscure. The lack of information gave ample scope for speculation and controversy, and there was some support for Beijerinck's conclusion that viruses were novel kinds of infective agent, but most people were for long content to assume they were ultramicroscopic organisms. What evidence there was certainly seemed on their side. The established facts were that viruses multiplied readily in susceptible organisms and, in doing so, usually produced offspring whose pathogenic behaviour was indistinguishable from the parent type, though sometimes, and especially when propagated in a new host or environment, they gave rise to variants with other properties. Opposers of the idea that viruses were organisms had nothing definite to balance against these two prime characteristics of living organisms, the ability to multiply and to vary. Small size and obligate parasitism were easily dismissed, for some organisms are only just visible with the microscope and some quite sizable ones will not grow on nutrient agar. Also, it seemed logical to correlate small size with obligate parasitism; after living inside cells where all the necessary food exists ready made, a bacterium might well get lazy, cease to make its own food, and ultimately lose the mechanisms for doing so.

This concept of viruses deriving from bacteria by a progressive loss of form and function achieved considerable popularity; it certainly was more plausible than a rival theory that viruses represented upward stages in the evolution of living organisms from inanimate matter, for the inability to multiply except in living cells is hardly a property that would favour the survival of the first forms of "life." Of other theories that were advanced we need note only one, the "free-gene" theory, with its origin based on resemblances between some genetic abnormalities and virus diseases, and which suggested that viruses were genes that had escaped from the chromosomes and were acting as independent entities.

VIRUSES AS NUCLEOPROTEINS

One consequence of considering viruses to be invisible organisms was that their study long remained almost exclusively the prerogative of bacteriologists and mycologists, who made great advances in many directions, but whose main techniques of microscopy and *in vitro* cultures were ill adapted to disclose the nature of invisible entities unable to multiply on artificial media. Not until the 1930s, when the techniques of protein chemistry were increasingly applied to the subject, was any direct information gained about the constitution of viruses. It was then found, first with tobacco mosaic but soon also with some other virus diseases of plants, that infected plants contained specific nucleoproteins. Were these the viruses? Or were they side effects of infection, products of the diseased host? Here is no place to review the immense amount of work done to try to answer these questions, and it will suffice to say that, with minor qualifications to be made later, nothing found in the last twenty years has conflicted with the conclusion that these nucleoproteins *are* the virus particles.

The properties of purified preparations of these plant viruses contrast greatly with those of even the smallest bacterium. Not only do many of them form crystals when precipitated appropriately, so indicating a uniformity of particle size that would be unusual with organisms, but X-ray analysis shows that each particle has a regular internal structure, with its component units arranged in a fixed manner, very different from the continuously changing arrangements characteristic of the insides of living cells. Then, too, the virus particles are chemically so much simpler; they have only two constituents, protein and nucleic acid, whereas even the simplest micro-organism contains, in addition to these two, a great range of substances of many sizes and chemical constitutions. Indeed, in their constitution, their apparent inertness *in vitro*, and their behaviour when subjected to various physico-chemical tests, these viruses resemble single components of cells rather than whole cells, and their nearest analogues seem to be the nucleoproteins that occur in every type of normal cell.

As chromosomes are also largely nucleoprotein and genes are carried by the chromosomes, at first sight there might seem reason for resuscitating the "free-gene" theory of viruses. However, although there are undoubtedly more useful analogies to be drawn between virus infections and genetic phenomena than between virus infections and the preying of one organism on another, this theory will not stand up to close examination. Virus particles are more complex than single genes, for there is much evidence that a virus has many characters that can be inherited independently, and that different strains of one virus when multiplying together can interchange characters and yield "hybrid" progeny. If a comparison is to be made, then it is to be between a virus particle and a collection of genes, or a piece of a chromosome, rather than with a single gene; but the chemical evidence makes even this comparison inappropriate.

The nucleic acid characteristic of the chromosomes has for its sugar deoxyribose and for its purine and pyrimidine bases adenine, guanine, thymine and cytosine, whereas the nucleic acid in all the viruses from higher plants that have yet been examined contains the sugar ribose and has uracil as a pyrimidine base instead of thymine. In this respect these viruses resemble the nucleoproteins of the cytoplasm rather than those of the nucleus. Indeed, there is nothing in the gross chemical constitution of these viruses that distinguishes them from the cytoplasmic nucleoproteins; their analyses show no new kinds of amino-acid in the protein moiety, or different sugar or purine and pyrimidine in the nucleic acid. Their peculiar ability to infect and to multiply in cells of susceptible organisms apparently derives from the way in which their component units are arranged to compose the virus particles, rather than in any peculiarity of the units. As the same kind of building units might lead to a cottage, cinema or church, depending on their number and the way they were put together, so it seems the same units can be used to make either "normal" nucleoproteins or virus particles.

Although all the viruses yet examined from higher plants contain nucleic acid of the ribose type, this is not true of all viruses. Those that infect bacteria and some of those that infect insects contain deoxynucleic acid; at least one of those that infects bacteria—bacteriophage T_2 of *Bacterium coli*—is also chemically different from usual deoxynucleic acid, for it contains 5-hydroxymethylcytosine instead of cytosine, but this is the only virus in which as yet any novel constituent has been identified. With tobacco mosaic virus the search has been so complete, and so much of the constitution is already accounted for, that further components are highly unlikely to be found. Many strains of this virus have been hydrolysed and analysed for their content of individual aminoacids, purines and pyrimidines. Strains that differ considerably in their pathological behaviour usually have different aminoacid constitutions, sometimes to the extent that one strain may contain an aminoacid not present in another but usually differing only in the amounts of some aminoacids. All seem to have threonine as a terminal aminoacid, which can be removed by carboxypeptidase without affecting the infectivity of the virus particles. The nucleic acid in different strains shows no comparable differences and the proportions of the purines and pyrimidines in 15 strains that infect solanaceous hosts are all similar, although there are differences in the adenine and uracil contents between these and strains that infect cucurbitaceous plants.

Different individual viruses have characteristically different compositions. The percentage of nucleic acid varies from 6 with tobacco mosaic and potato virus X to 18 with tomato bushy stunt and 40 with tobacco ringspot virus, and the nucleic acids of different viruses also have distinctive proportions of individual purines and pyrimidines. Some have them in similar proportions to the prototype of ribose nucleic acid, that from yeast, but others differ widely from it; for example, potato virus X is unusually rich in adenine and turnip yellow mosaic virus in cytosine, whereas southern bean mosaic contains these two and uracil and guanine all in approximately equal amounts. In normal cells, ribose nucleic acid is thought to be specifically concerned in protein synthesis and in determining the identity of protein synthesized, and the wide differences between the aminoacid constitution of different viruses is probably correlated with these differences in the nucleic acid. Other than this, it cannot be said that the detailed analysis of the composition of viruses has been helpful biologically, and it is not possible to relate any of these quantitative differences in composition to specific properties of the viruses.

THE MORPHOLOGY OF VIRUS PARTICLES

Early studies on the physico-chemical properties of purified preparations of plant viruses made it evident that some, such as tobacco mosaic virus and potato virus X, which form liquid crystals and show many anomalous physical properties, had rod-shaped particles, and that others, such as tomato bushy stunt and tobacco necrosis viruses, had particles that were approximately spherical and of a uniform size. The development of the electron microscope allowed direct observations on particle sizes and shapes of virus particles and confirmed the earlier conclusions.

The plant viruses so far examined have fallen into three main types: one, characterized by potato virus X, is a long, flexible, fibre-like particle (Fig. 1); the second, typified by tobacco mosaic virus, is a rigid rod (Fig. 2); the third, typical of viruses that form true crystals, is approximately spherical (Fig. 3). The first and second types have uniform widths—about 12 mμ* for potato virus X and 15 mμ for tobacco mosaic virus—but they occur in a range of different lengths; many treatments make them aggregate end to end, although one length is usually found more commonly than another, e.g. about 300 mμ for tobacco mosaic virus. Many of the spherical viruses are about 25 mμ in diameter and some are as small as 17 mμ. Whether these particles are truly spherical is doubtful; they are more likely to be polyhedra, but there is yet no unequivocal evidence of plane surfaces with these small particles.

The bacterial viruses have more entertaining shapes; many resemble miniature tadpoles, with very distinctive heads and tails (Fig. 6). Some of these are much larger than the viruses from higher plants we have been considering, and there seems no doubt that their heads are not spheres but polyhedra. It is also clear from many lines of work that they are not uniform masses of nucleoprotein, but that the nucleic acid is carried inside

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* 1 mμ or millimicron = 10^{-6} mm.

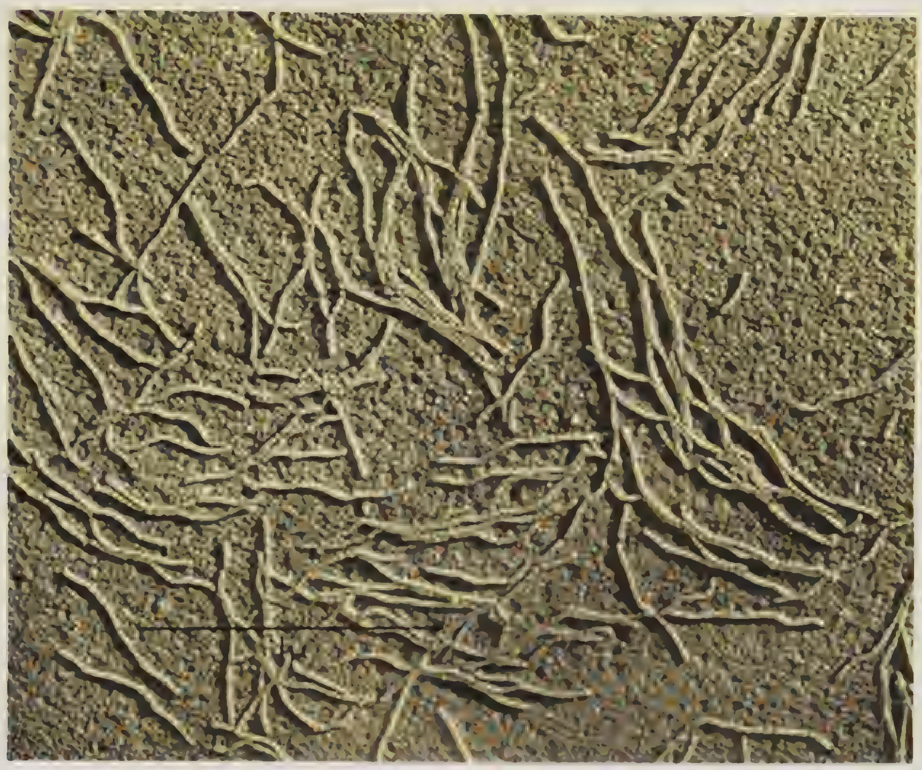

FIG. 1. Electron micrograph of potato virus X in clarified infective sap, showing the narrow, flexible particles. (By permission of Rothamsted Experimental Station.)

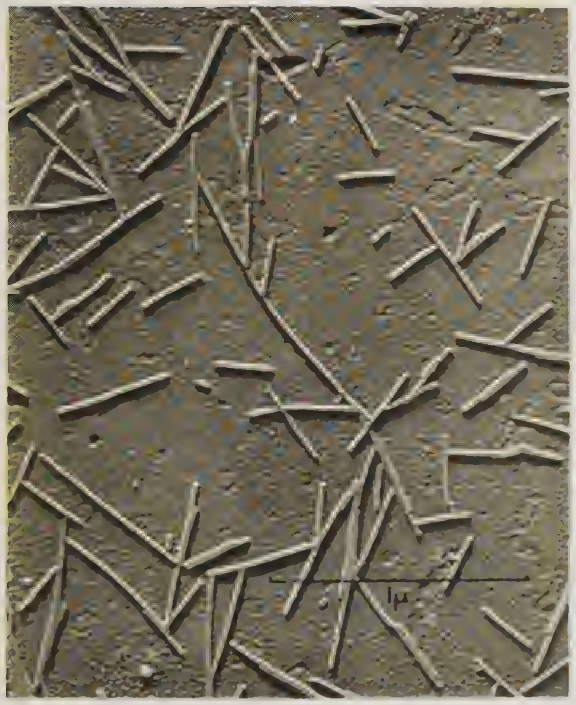

FIG. 2. Electron micrograph of purified preparation of tobacco mosaic virus, showing rigid rod-shaped particles of differing lengths. (By permission of Rothamsted Experimental Station.)

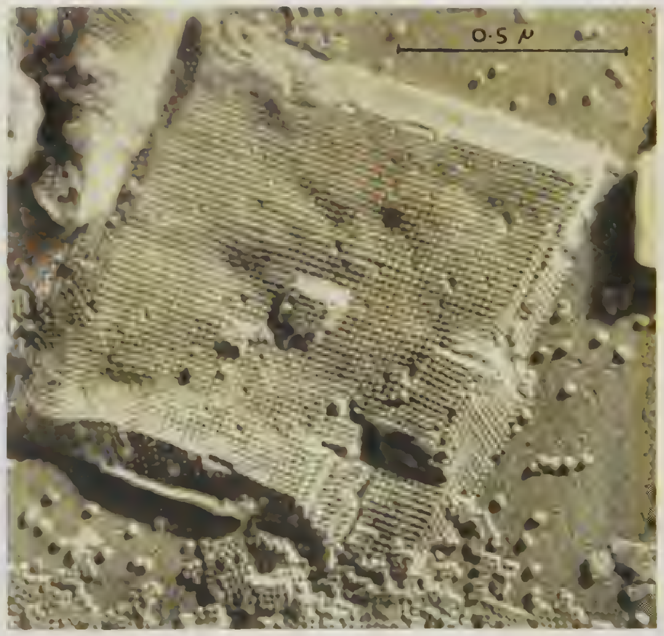

FIG. 3. Electron micrograph of a carbon replica of a small crystal from a purified preparation of the Rothamsted culture of tobacco necrosis virus, showing the arrangement of the spherical virus particles in the crystal. (By permission of Dr. R. W. G. Wyckoff, National Institute of Health, Bethesda, U.S.A.)

the head, the outside of which is protein, as also is the tail. Although their shapes are so different from those of plant viruses, and they contain a different type of nucleic acid, the arrangement of their two components may not be entirely dissimilar. There is for example good reason to think that turnip yellow mosaic virus may have particles that resemble the heads of bacteriophages inasmuch as the nucleic acid is carried inside a protein shell. Plants infected with this virus yield two types of specific particle, both of much the same size, with the same electrophoretic mobility and seemingly with the same antigenic constitution. Only one of these types, however, contains nucleic acid; the other, which is lighter and is not infective, seems to be the same protein but minus the nucleic acid. As such properties as electrophoretic mobility and antigenicity are likely to be determined by the surface of particles, here is strong suggestive evidence that the surface is protein only.

With the rod-shaped tobacco mosaic virus, too, there is evidence from several lines of work that the nucleic acid is localized within the protein. It came first by fragmenting the virus by various treatments and examining the fragments with the electron microscope. Treatment with alkali separates the nucleic acid from the protein and gives a succession of protein fragments, down to those with a weight equivalent to a molecular weight of 17,000, which seem to be the sub-units from which the protein is built. When only partially degraded, to give fragments containing about 10 of these sub-units, discs of the width of the virus particle are sometimes found with holes in their centres. No holes are seen in otherwise similar discs produced by ultrasonic disintegration of the particles (a method that does not separate the nucleic acid from the protein), and it seems that the central holes in the alkali-produced discs indicate the original site of the nucleic acid. Small protein particles free from nucleic acid also occur in sap from infected plants, and these too have holes in their centres. When the virus is disintegrated with alkali, thin, fibrillar material can sometimes be seen with the electron microscope, and this is presumably the nucleic acid (Fig. 5). The position of

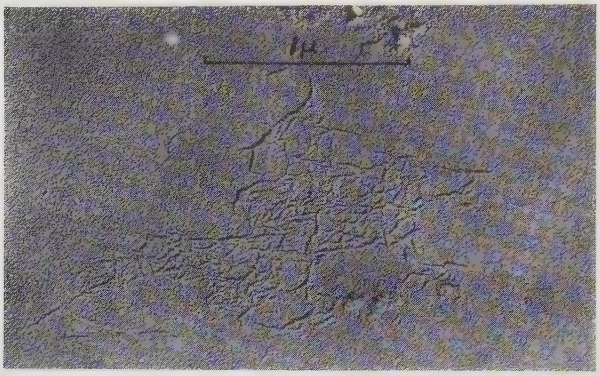

FIG. 4. Electron micrograph of the nucleic acid of tobacco mosaic virus, separated from the protein by phenol. Such preparations have about one-hundredth the infectivity per unit of nucleic acid of normal virus preparations such as pictured in Fig. 2. (By permission of Rothamsted Experimental Station.)

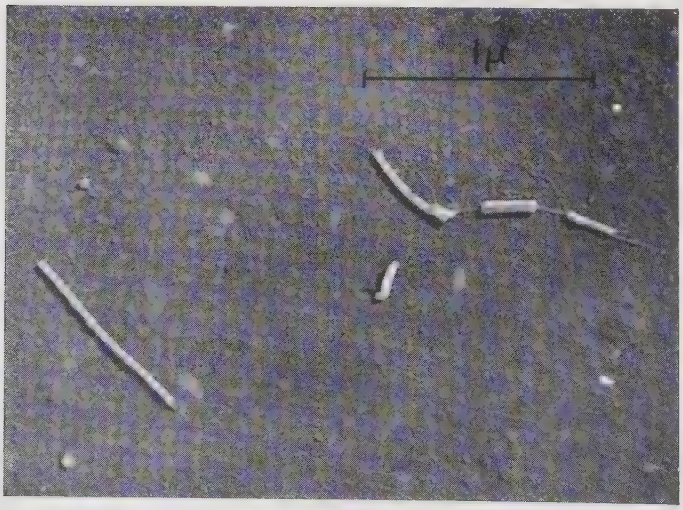

FIG. 5. Electron micrograph of partially degraded tobacco mosaic virus, showing fibres, presumably nucleic acid, joining pieces of intact rod and protruding from the ends of rods. (By permission of Rothamsted Experimental Station.)

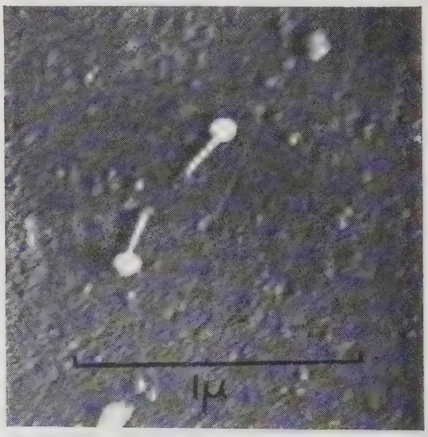

FIG. 6. Electron micrograph showing the tadpole-shaped bacteriophages that attack *Rhizobium* sp., the bacteria that produce nodules and fix nitrogen in roots of leguminous plants. (By permission of Rothamsted Experimental Station.)

the nucleic acid has been shown more definitely by disrupting the virus particles with a detergent, which appears to dissolve the protein from the ends of particles. When done appropriately, the partially degraded rods have central fibres protruding from the ends of the shortened particles. Treating such preparations with the enzyme ribonuclease, which hydrolyses ribose nucleic acid, removes the protruding fibres.

X-ray determinations of the radial density of tobacco mosaic virus likewise suggest that the nucleic acid occurs deeply embedded in protein. These show that a particle is not a solid rod, but a tube with a hole of 2 mμ radius up the centre. Lining the tube is a layer 2 mμ thick of low density, and then at 4 mμ from the centre is a region of high density. As the phosphate groups in the nucleic acid effectively scatter X-rays, this indicates that the phosphate–sugar chain of the nucleic acid lies at a radial distance of 4 mμ. That this is the position of the nucleic acid is further indicated by measurements on particles free from nucleic acid. When the protein and nucleic acid are separated from one another with alkali, the protein can on its own be made to re-aggregate, or re-polymerise, to form rods, which in the electron microscope look like virus particles. These rods give X-ray patterns that resemble those of the virus particles in some respects, but there is one large difference: instead of a density maximum at a radius of 4 mμ there is now a density minimum. Outside the zone of the nucleic acid, less dense X-ray scattering material extends to a distance of about 9 mμ from the centre.

The X-ray patterns suggest a diameter of about 18 mμ for the particles of tobacco mosaic virus, which is 3 mμ more than when aligned, packed particles are measured with the electron microscope. It is, though, about the width obtained when isolated particles are measured under the electron microscope. The X-ray diffraction patterns suggest an explanation for this apparent anomaly. They show that the sub-units of protein, like the nucleic acid, are arranged spirally round the long axis of the particle, probably with 49 of them for every three turns of the spiral, and that the surface is deeply indented with spiralling grooves. The external appearance, therefore, could resemble that of a rope wound round a cylinder. The grooves allow neighbouring parallel particles to interlock, so that the average diameter when calculated from electron micrographs of closely packed particles is less by the degree of interlocking than the total distance across individual particles.

VIRUS MULTIPLICATION

For a time after plant viruses were first isolated as nucleoproteins these were thought to be the minimal infective requirement and to be self-reproducing, but this idea has now to be abandoned. Its fallacy was first shown from studies on bacteriophages, with which it was found that the tail and the protein coat of the head remain outside infected bacteria. The bacteriophage attaches itself tail first to a susceptible bacterium and seems to dissolve a hole through the wall, through which the nucleic acid from the head and little if anything else then passes into the bacterium. But this is enough, and in due course new bacteriophage particles, complete with coats and tails, are formed and dissolve the host cell. Clearly the tail and the protein coat are not self-reproducing systems: they do not even enter the cells from which new ones later emerge. By contrast the nucleic acid appears to be more than self-reproducing, for its entry leads to the production of more of itself and of its characteristic protein trappings.

How far this is true with other viruses is, as yet, uncertain, but recent work with tobacco mosaic virus suggests that the nucleic acid alone is enough to initiate infection and lead to the production of more virus. Various methods can be used to separate the protein from the nucleic acid of tobacco mosaic virus. All of these greatly decrease infectivity and most of them denature the protein. Exposure to alkali in the cold, however, produces soluble protein fragments which, when made acid, re-aggregate to form long rods resembling the original virus particles. If re-aggregated in the presence of nucleic acid, the re-formed rods may also contain the 6% of nucleic acid characteristic of virus particles. Hence the physical manufacture of structures resembling virus particles can be achieved *in vitro* from smaller units. Indeed, it has been claimed that infective particles can be produced *in vitro* by combining non-infective components, but this was probably an error of interpretation before it was realized that nucleic acid alone can be infective. Its infectivity was shown by disrupting the virus with phenol or detergents, which denature the protein and give nucleic acid preparations that are certainly free from intact virus particles and contain no demonstrable protein (Fig. 4). In this state the nucleic acid is only a hundredth or less as infective per unit of nucleic acid as the intact virus, and it is also very unstable. Whereas tobacco mosaic virus remains infective at room temperature for years and is unharmed by exposure to ribonuclease, the nucleic acid preparations lose their infectivity in a matter of hours, and in a matter of minutes when exposed to the enzyme. Re-combining the nucleic acid with the protein stabilizes it, and unless experiments are carefully controlled such stabilization can be misconstructed as creating infectivity. Another complicating factor in such experiments is that the protein fragments produced by disrupting the virus with alkali, like many other proteins, are inhibitors of infection, and high concentrations of them can obscure the presence of infective nucleic acid or nucleoprotein.

Although disrupting and reassembling particles of tobacco mosaic virus may lack all the drama and glamour that would attach to making infective virus from non-infective components, it is in itself a major and significant achievement. It suggests how the particles may be put together in cells and, considered together with results from studying bacteriophages, gives us some kind of picture of the different roles of the two components and of the processes concerned in infection and virus multiplication. It is clear that the protein of the bacteriophage not only protects the fragile nucleic acid from inactivation but also provides the mechanisms whereby the nucleic acid gets from one cell to another, dissolving a small hole to let it in and bursting the whole bacterium to let the tadpoles out.

Biochemical studies and examination of artificially broken bacteria with the electron microscope further show that the first effect of infection is to swing the nucleic acid synthesis of the bacteria over to making deoxynucleic acid; at about half the time between infection and bursting of the bacteria, protein particles that are serologically related to the bacteriophage begin to form and their formation is followed by the appearance of the characteristic tadpole-like particles, which increase in number until the bacterium bursts. The specific nucleic acid and protein are apparently first produced separately and then brought together to give the completed virus particle.

The action of the protein of tobacco mosaic virus in protecting its nucleic acid from inactivation has already been mentioned, and it may have other activities, such as aiding the entry of the nucleic acid into some specific cell component or causing the changes that show as the characteristic symptoms of infection. There is no positive evidence for these other activities, but the ability of the protein to inhibit infections is perhaps a pointer that it may engage in some first step of infection which aborts because there is no nucleic acid to take the process further. For example, if the first step in infection by normal virus particles is for the protein to attach itself specifically to some host cell component, as a preliminary to releasing the nucleic acid, this attachment could be prevented if the site were already occupied by protein free from nucleic acid. It must be stressed that there is no definite evidence that infection with plant viruses entails separating the nucleic acid and protein of virus particles, but that it may is suggested by the fact that, shortly after they are inoculated to leaves, the particles enter a temporary unstable phase during which infection can be prevented from becoming established by treating the leaves with ribonuclease.

As with the bacteriophage, the protein and nucleic acid that go into tobacco mosaic virus seem to be synthesized separately in the host cells. When extracts from infected leaves are centrifuged at high speed, the rod-shaped particles sediment, but the supernatant fluid still contains some smaller proteins that are serologically related to the virus protein. These contain little or no nucleic acid and also resemble the protein fragments produced when the virus is disrupted with alkali in their ability to aggregate and form rods of the size and shape of virus particles. The results of experiments in which infected leaves were fed with radioactive isotopes suggest that these small proteins are not breakdown products of virus particles but transient products of synthesis that are soon transformed into some more permanent substance. The almost inescapable conclusion is that they are intermediate stages in the formation of the virus protein, able to join together and with nucleic acid, as can the alkali-produced fragments *in vitro*, to give the characteristic rod-like virus particles.

Our picture of infection and virus multiplication is still no more than a bare outline sketch, with all the detail waiting to be filled in. Yet, however inadequate it may be, it does show something very different from the earlier picture of virus diseases as the equivalent of one organism preying on another, with viruses behaving like bacteria, growing on food taken from the host, and multiplying by binary fission. It is not only that viruses are simpler than organisms and can be subjected to indignities, such as being fragmented and reassembled, which no bacterium would tolerate, but they have very different and much more intimate relations with their host cells. In effect they become part of the host cell and depend on it for the substances from which they are made, and also apparently for the mechanisms whereby they are made. For long it was the practice in horticulture to give certain virus-infected plants, for example variegated tulips, varietal names different from those of their uninfected forerunners. In doing this horticulturists were in a sense anticipating the conclusions of recent research, which is that infection adds new potentialities to the host cell, giving a result equivalent to that of mutation or of crossing with a genetically different type, and most simply described as turning one variety into another.

In normal cells the quality and quantity of protein made seems to be determined by the quality and quantity of the nucleic acid. Susceptibility of a cell to a virus may reflect the ability of the virus nucleic acid to combine or associate successfully with the nucleic acids already present. The whole infected cell is the biologically active unit, and it seems a misnomer to call the virus particles self-reproducing; the nucleic acid may be, but the cell contains the mechanisms for making aminoacids, purines and pyrimidines and for building them into proteins and nucleic acids. The intrusion of the virus nucleic acid into the cell brings a new pattern, or adds a new set of instructions, to the building process and leads to a new set of end products, some but not all of which are infective virus particles. To some cells the derangement of the metabolism is lethal, to others damaging though bearable, whereas some cells can accommodate it without appearing to be harmed.

CONCLUSION

With this concept of virus diseases we can go far to reconcile the differing behaviour of viruses *in vivo* and *in vitro*, aptly summarized by the past conflicts between those who have called viruses organisms and those who have called them molecules. In the test-tube they are inert, and although they are too complex and variable ever to have warranted being called molecules, their behaviour does not differ appreciably from that of other nucleoproteins. In susceptible cells they seem to come alive, but their vitality is not solely theirs, and their apparent ability to multiply and mutate reflects the activities of the whole living cell, of which they are now one component determining what goes on.

Does the nature of viruses matter? Has it any practical importance? For such problems as controlling virus diseases by vaccines, by eradicating sources of infection or by killing the vectors that spread them, it probably matters little whether viruses are organisms or not, but for others it matters a great deal. There are now antibiotics or other drugs for curing most bacterial diseases of man and animals, but it is significant that there is none for curing virus diseases. This considerable difference can be attributed to the fact that, whereas bacteria have independent synthetic activities, which can be specifically interrupted by drugs, viruses do not.

Knowledge of the constitution of viruses suggests that substances most likely to interfere with their multiplication are those that will affect the synthesis of nucleic acid, and some analogues of purines and pyrimidines are already known to decrease the multiplication of tobacco mosaic virus. It is not surprising that these also affect the host plants deleteriously, for if viruses use the same building mechanisms and the same building bricks as are used for making the host's nucleic acid, it asks a high degree of specificity of something that it should stop one and not the other. Such substances may be found, either empirically or because of growth in knowledge about the structure of different nucleic acids, and the search for therapeutic substances for other virus diseases may be easier than for tobacco mosaic.

The ideas and conclusions summarized in this article derive largely from work on a few plant viruses and bacteriophages. The similarities in their behaviour certainly suggest that many other viruses will also behave in much the same way, but to extrapolate from these to all others is dangerously premature. Some may be chemically more complex, have novel components or more independent activities, and, if so, therapeutic treatments will be correspondingly easier to find. Having said this, however, it must be added that as increasingly more viruses are studied chemically, so the number known to be nucleoproteins increases and the less likely it seems to become that any are not, although it can safely be assumed that there are many differences between their individual nucleic acids and proteins and the ways in which the two are put together.

DEVELOPMENTS IN THE FIELD OF PARASITIC BRONCHITIS

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Rapid progress has now been made in the control of lungworm disease. An anthelmintic has been found that is effective against adult worms, and a devitalized larval vaccine at present on field trial is showing promise of safely conferring immunity to the disease. How animals acquire resistance is well understood, but husbandry measures have not been devised that would enable grazing stock to be raised in relative immunity to infection.

In the course of the past ten years a growing amount of research has been devoted to the study of parasitic bronchitis or lungworm disease of farm animals, with interesting and important results. A few of these discoveries have already passed the experimental stages and are now beginning to be applied in practice; but knowledge of what has been happening is still largely confined to research workers, and it is time that it reached a wider public. This article will outline the nature of the lungworm problem and discuss the new developments and their practical significance.

IMPORTANCE OF THE DISEASE

The chief characteristic of lungworm disease, usually called "husk" or "hoose" by the farmer, is that the worm parasites in the lungs and air passages of affected animals provoke an inflammatory reaction that is frequently severe enough to cause respiratory distress and sometimes death. Secondary infections of the lung with bacteria and viruses may supervene. The disease is mainly seasonal, occurring towards the end of summer in temperate countries, and outbreaks vary in severity from year to year. Until recently no specific drug against lungworms has been available, and control of the disease has been limited to methods of husbandry designed to interfere with the development of severe infestations in susceptible stock.

As with other animal diseases, it is impossible to arrive at reliable figures for the economic losses caused by lungworm infestation. The importance of a disease of livestock is measured not only in terms of the animals it kills but also by the depressing effects it may have on growth and production; allowance must be made for the effect it may have on the management of the farm in respect of the replanning that may be required to replace affected animals; and diseased animals often require, over and above therapautic treatment, extra care and nursing, which means that both feed and labour must be diverted from other needs on the farm. From all these points of view parasitic bronchitis is an important disease, especially in cattle.

Statistical data on losses are invariably inadequate and usually absent. Nevertheless, figures from such surveys as have been made give a rough idea of the damage. In Great Britain an estimate of £3,000,000 per annum has been made for lost animal production due to parasitic bronchitis among cattle, sheep and pigs. One-half of all bovine deaths during August to October 1955 has been attributed to husk [7]. An outbreak in a herd of 35 milking cattle with only one death cost the farmer £2500, mainly in loss of milk and extra food [12]. The incidence of the infection in pigs in Britain has been variously estimated at between 20% and 47% [3, 22, 24] and that in sheep is believed to be at least 90%, although the morbidity figure for this animal is particularly misleading, because the commonest worm (*Muellerius capillaris*) is of low pathogenicity. Figures for countries other than Britain are scarce. In the U.S.A. a small-scale experiment involving infested and control calves showed a 10% weight difference 70 days after infection [19]. 70% of pigs in the United States are infested [1]. The disease is common and often severe in goats and sheep in the countries bordering the Mediterranean, and in cattle in north-west Europe.

THE LUNGWORMS

Lungworms are found in most countries from arctic to tropical regions, and the various species infest animals as diverse as seals and shrews. Here only a few of the most pathogenic to domestic animals will be referred to in any detail. These belong to the family *Metastrongylidae* and to the same order, *Strongyloidea*, as the common intestinal nematodes.

Dictyocaulus

D. filaria (in sheep and goats) and *D. viviparus* (in cattle) are probably the most pathogenic lungworms and have a world-wide distribution. In several respects these species vary slightly from each other, but their main biological features are closely comparable and one description will, in general, cover both.

The adult worms are creamy-white, threadlike, and 3–10 cm. in length (Figure 8). They inhabit the bronchioles and bronchi, living on exudate produced there (Figure 5). The embryonated eggs hatch in the bronchi, or in the digestive tract when they are swallowed. The larvae are passed in the faeces, move on to pasture herbage, and, after being swallowed by a suitable host, migrate to the lungs. During this complex progress the larvae undergo several changes which require more detailed description.

First-stage larvae in the faeces moult under favourable conditions into the second and third stages, all living on a contained food supply. The time required for these changes may be as little as four or five days or as long as six weeks in cold weather. These third-stage larvae are infective (Figure 7). Unlike larvae of the preceding stages, which are not particularly hardy, they can resist freezing for a fortnight or more [17]. In moist, cold climates they have been known to survive on pasture for as long as eleven months, but in dry climates they usually die within one or two months; desiccation appears to be the principal lethal environmental factor.

Infective larvae migrate up pasture plants in water films but, when the dew goes off the grass, return to a more humid environment at soil level. Thus pasture is most dangerous in warm, humid weather, and at night. Pasture concentrations of larvae tend to be lowest in winter and highest in late summer and autumn, and because the number of infective larvae on pasture at any given time depends more on climatic factors than on the number being passed in the faeces, even lightly infested animals may produce a lethal pasture contamination under favourable conditions.

Third-stage larvae can live in water in the margins of ponds and puddles, so that animals drinking at such places may be infected, though drought conditions prevail on the pasture.

The survival of larvae in "cowpats" depends on the degree of dehydration that develops; should the outer surface bake dry, larvae will not escape to contaminate the pasture, and in fact they have little power to migrate from even moist faeces. Consequently the spreading of dung by grazing stock, harrowing, or other means increases the risk to grazing animals. Larvae in loose diarrhoeic faeces scattered on well-grown herbage rapidly build up lethal infestations [18]; such circumstances frequently obtain under modern conditions of dairy farming, and the use of a back-fence in strip-grazing is thus particularly important.

When infective larvae are swallowed they traverse the bowel wall and pass into the mesenteric lymph glands approximately four days after ingestion, at which point the fourth larval stage is reached. In about six days the larvae enter the vascular system and are arrested in the lung capillaries. In the lungs they develop into a fifth stage and break through the alveolar walls into the alveoli and bronchioles, where they attain the adult reproductive stage. The cycle takes about three weeks from the time of infection. Experimentally, infective larvae have been recovered as early as 23 days after infection.

Adult worms may persist in the host for a considerable time, perhaps several years [22]. Infestation survives the winter in carrier animals. Naturally infested lambs have been shown to eliminate larvae for 127 days [5].

The phenomenon of "inhibited development," recognized in the intestinal worms, occurs also in lungworms of this genus. Fifth-stage larvae have been found in the lungs of sheep and cattle several weeks or months after natural and artificial infection, and these retain their power to develop after the elimination of any adult worms present [21].

Protostrongylus and Metastrongylus

Worms of these genera differ from *Dictyocaulus* spp. mainly in requiring intermediate hosts to complete their life cycles.

Protostrongylus rufescens is a widespread parasite of sheep, goats, and some other herbivores. The worms are about 1½–3½ cm. long and reddish-brown in colour, and their life cycle involves certain terrestrial molluscs. The adults live in the small bronchioles.

Three species of *Metastrongylus* infest pigs. All have similar life cycles, with earthworms as intermediate hosts. The following description is based on what is known as *M. apri*.

Embryonated eggs are laid in the bronchioles of the pig, pass into the trachea, and are swallowed. For their further development the first-stage larvae or embryonated eggs appearing in the faeces must be swallowed by earthworms, and about seven to ten days after this has happened the larvae reach the third stage, which is infective for pigs. Very large numbers of larvae—as many as 2000—are sometimes found in a single earthworm; yet the intermediate host evinces little discomfort and has been shown to retain the infestation for as long as 32 months [Shope, cited by Lapage].

Pigs become infected by eating infested earthworms. The larvae follow a similar path to that taken by the *Dictyocaulus* species, the third moult taking place in the mesenteric glands and the fourth and final moult in the lungs. Egg-laying begins about 24 days after infected larvae are ingested; peak egg output follows in a few days and is maintained for three to five weeks.

PATHOLOGICAL ASPECTS

Effects on the Lungs.

In recent years much work has gone into the pathological examination of experimentally and naturally infected animals [3, 8, 10, 11, 19]. Three principal effects of the worms in the lungs have been recorded.

1. When the invading larvae break through the pulmonary capillaries into the alveoli they provoke a marked cellular reaction, and the profound changes that ensue may lead in severe cases to oedema of the lung (Figures 3, 5) and heart failure.
2. As the adult worms develop, bronchial exudate and cellular detritus may block completely some bronchioles. Further, some of the eggs and newly hatched larvae produced at this stage are inspired into the ventilating areas of the lung, causing an intra-alveolar reaction. Escape of air from ruptures in the lung tissues may cause swellings under the skin.
3. Solidification of affected regions of the lung precedes natural recovery.

Secondary infection may intervene in the second or third stage, seriously diminishing the chances of recovery of the affected animal.

Clinical Signs

Where the disease begins mildly, some coughing will be heard. This sign, often held to be a diagnostic feature, is absent in severely affected animals. Other outbreaks may be heralded by loss of condition and fall in milk yield, followed by sudden deaths. Rarely, if ever, are individual animals infected: infestation is of the group, and where one animal is obviously affected some degree of infestation will be manifested sooner or later by others grazing with it.

After the initial stages of an outbreak a large proportion of the group show rapid, shallow breathing. Not only the thoracic muscles but also the abdominal muscles are brought into each effort of respiration. Expiration is frequently accompanied by a grunt. The severely affected animal subjugates all its actions to ensuring an air supply, and its body assumes a characteristic posture with head and neck outstretched, mouth open, ribs arched and abdomen tucked up. Some nasal discharge and frothing saliva may be seen. Appetite and thirst appear to be lost, and rapid emaciation and dehydration follow. The temperature is elevated and the milk yield falls sharply. Pregnant animals may abort. The general condition of an infested animal is well shown in Figure 1.

A hyperacute condition similar to "fog fever" is seen in cattle and sometimes sheep. Although many such field cases are explainable in terms of a massive larval invasion of the lung, this condition probably has an allergic component in its aetiology, for death may occur before the worms reach the lung [10].

Outbreaks of husk are terminated naturally by the recovery of surviving animals over a period of three weeks to several months. The duration of this phase depends on the amount of lung damage, intercurrent bacterial, viral and parasitic complications, and the food supply.

The signs seen in sheep (Figure 2) and goats vary according to the species of worm. With *D. filaria* and, to a lesser extent, *P. rufescens* they are similar to those in cattle, and sometimes as severe. *Muellerius* infections, which are common, cause only slight clinical effects.

In pigs the most serious changes are seen in young animals. Adult worms in the bronchioles and bronchi cause irritation and the production of exudate. In older pigs it is common to find only residual infestations, with a few worms in the terminal bronchioles of the diaphragmatic lobes [3]. Pig keepers will be familiar with the milder type of disease characterised by an increased respiratory rate, "thumping" expiration, occasional coughing, and unthriftiness. These symptoms are

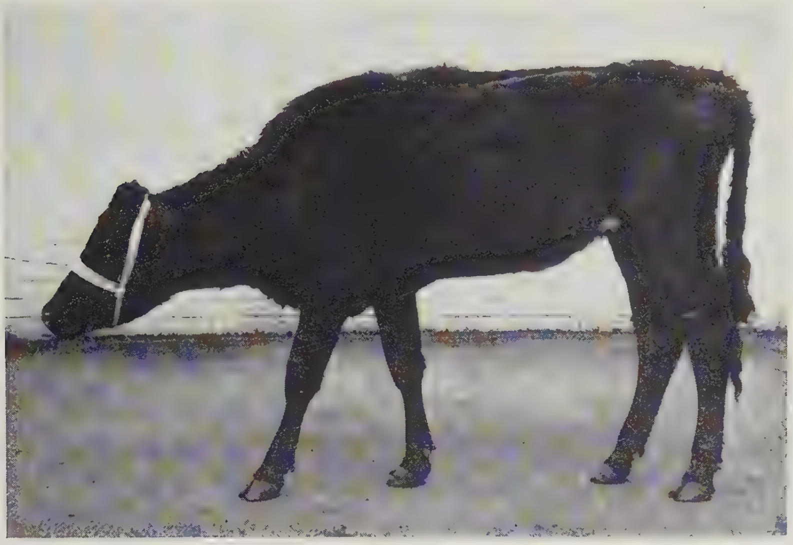

FIG. 1. Yearling suffering chronic infestation with lungworm.

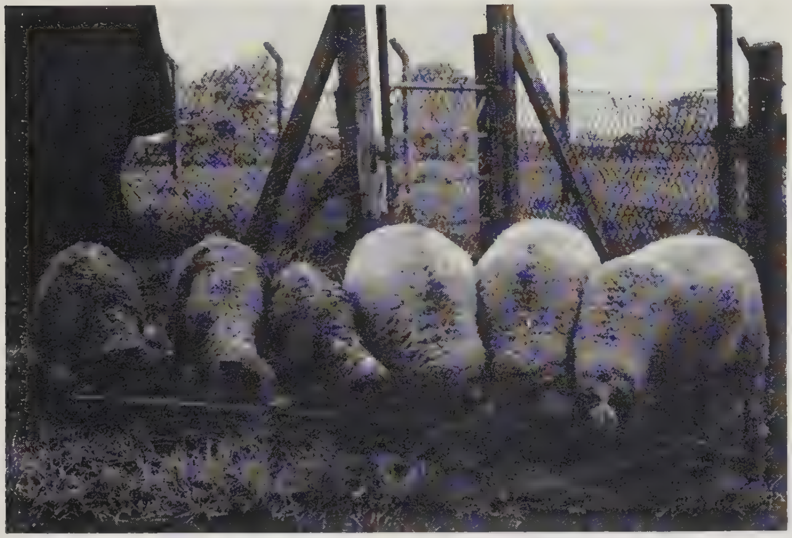

FIG. 2. Three sets of twin lambs, showing (trio on right) one of each pair raised worm-free, and the others (trio on left) reared on good pasture heavily infected with lungworm and intestinal worm larvae.

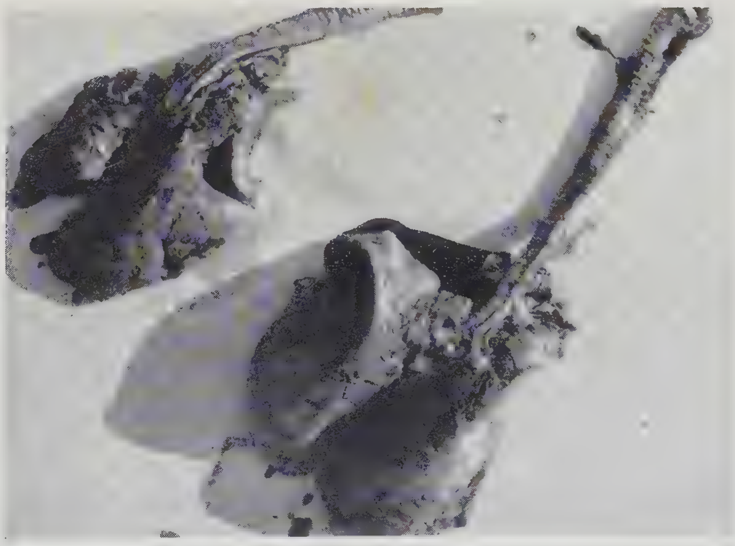

FIG. 3. Left: Normal lungs of goat. Right: Lungs of goat 21 days after infection with *D. filaria*. Oedema accounts for the difference in size of the two specimens.

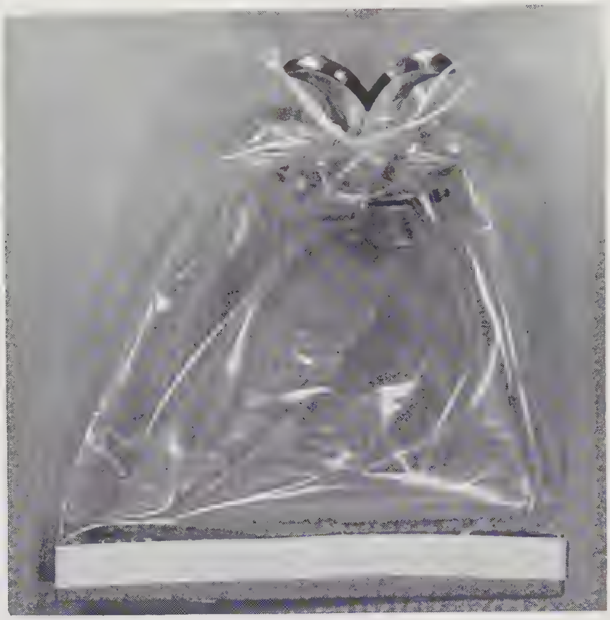

FIG. 4. Tracheotomy tube and polythene sac used for recovering worms and exudate from experimental animals.

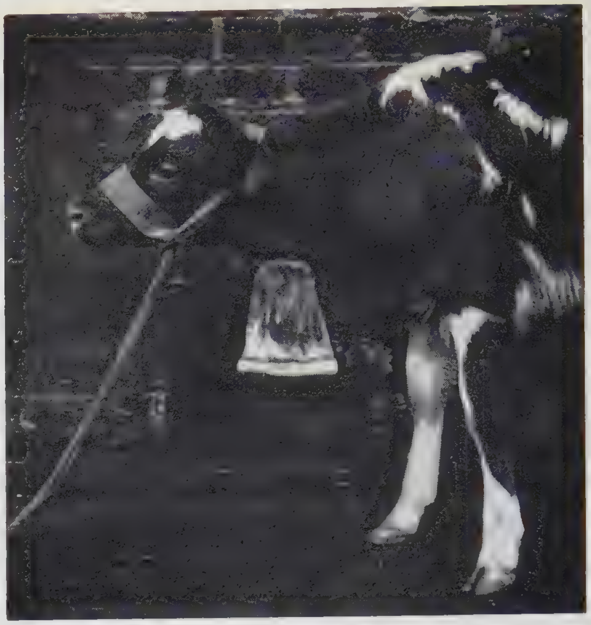

FIG. 6. Calf showing early signs of parasitic bronchitis, with tracheotomy tube and collecting sac in position.

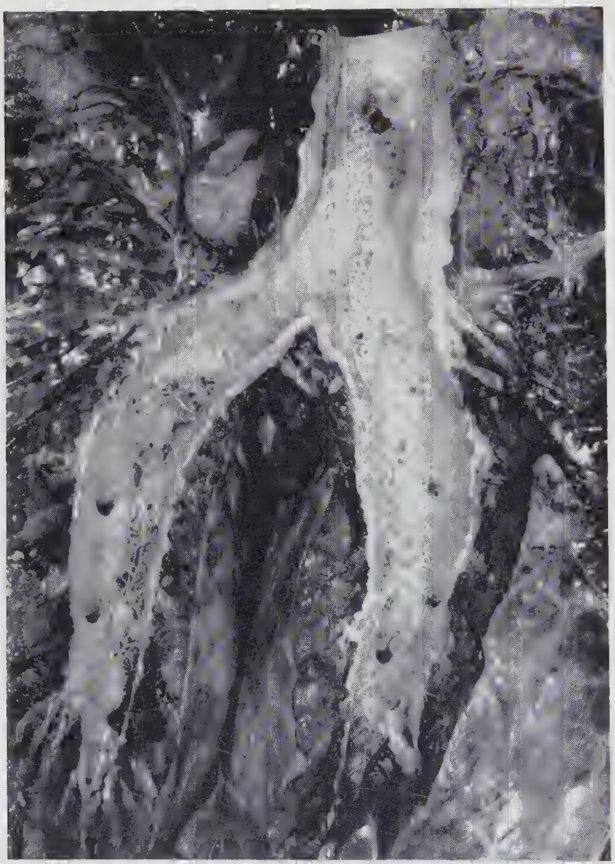

FIG. 5. *Dictyocaulus filaria* in the bronchioles and lower trachea. Note the oedematous nature of the lung.

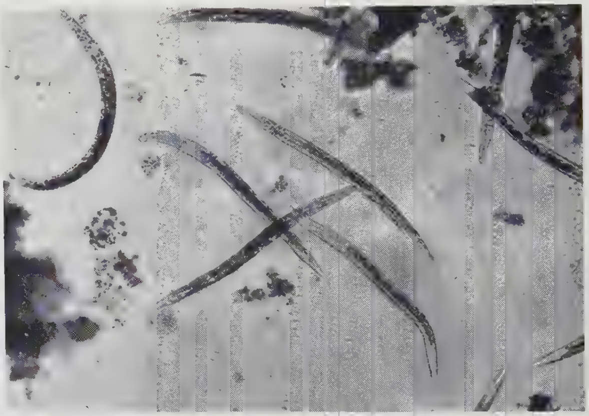

FIG. 7. *D. viviparus* infective larvae.

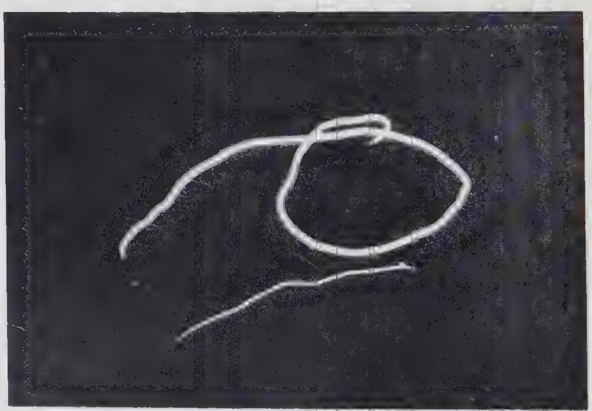

FIG. 8. Female (above) and male (below) of *Dictyocaulus viviparus*.

clinically similar to those caused by migrating ascarid larvae. Such pigs may recover spontaneously in a few weeks, though it commonly happens that one or two of each affected litter are slowed in their growth and remain affected with a chronic bronchitis. The residual infestation found in pigs of five to six months of age is probably harmless; but such pigs are a danger as carriers, and care must be taken to confine them to concrete pens and to spread their dung only on arable land.

Natural Immunity

All age groups of animals are susceptible to infection and acquire resistance only by the ingestion of infective larvae. Because the larval population varies so much thoughout the year from one field to another, considerable differences in the degree of resistance will be encountered. So far, no practical measure has been devised that will ensure freedom from husk.

The natural disease in young stock may be caused by the third (or even later) generation of worms developed in the season [7]. If young cattle do not pick up a lethal dose in the first five days of grazing on infective pasture, they rapidly acquire a partial immunity; even if grazing is continued on the particular ground where the concentration of larvae ("larval challenge") is building up steadily, immunity restricts the number of infective larvae that develop into adults. Other animals, previously unexposed, introduced at this stage to such a pasture may die quickly [20]. Should climatic conditions prove especially favourable to the maturation of third-stage larvae in the circumstances described, then a violent outbreak is likely to occur even among so-called "immunes." In adults this may not be true, for outbreaks are associated with the sudden intake of large numbers of larvae left by stock that previously grazed the pasture [13].

CONTROL AND TREATMENT

Changes in the methods attempted for overcoming parasitic bronchitis reflect the development of veterinary thought on diseases in general.

Originally treatment was based on the introduction of irritant or sternutatory substances into the repiratory tract, either by injection or inhalation. Such treatments often provoked violent coughing which dislodged a small proportion of lungworms in some cases, but they imposed a severe tax on the cardiac and respiratory function and accelerated the death of some animals. Attempts have been made to increase the effectiveness of the treatment by using an anthelmintic, and an aerosol machine delivering ascaridole is the most refined of these modifications [4]. Systematic treatment has also been tried, using drugs that destroy the larvae of filarial worms, though so far only diethylcarbamazine has shown any effect [15, 16], and that only under rather special conditions.

During the last five to ten years efforts have been made to elucidate the complex features of the biology of the parasite and the epidemiology of the disease, and surveys of lungworm infestation in various hosts have revealed much information of fundamental importance. Much of what is known today on the disease is derived from studies at the Veterinary Laboratory of the Ministry of Agriculture at Weybridge, the Glasgow University School of Veterinary Studies, and the University of Liverpool. The original purpose of much of this research was to formulate a system of stock management that would interfere with the host-parasite relationship in such a way that stock could be raised in relative immunity to infection; but little practical progress has been made, mainly because of the wide variations of circumstance and husbandry obtaining in the field.

Arising from research into the host-parasite relationship, a novel method of control is at present under investigation. Experiment has confirmed the field observation that a strong acquired immunity is derived from *D. viviparus* infection. This immunity can be assessed by complement fixation tests and measurements of the output of first-stage larvae from "challenged" animals. Some artificial immunity can be conferred by vaccination with a whole-worm antigen, but much more effectively by the oral administration of larvae.

It has been shown that, by irradiation, the larvae of *Trichinella spiralis* could be so devitalized that they were unable to complete their development. Working on the hypothesis that immunity to infestation is acquired during the period of migration through the mesenteric lymph nodes, a level of irradiation was devised that substantially prevented the development of *D. filaria* larvae in the lungs without materially inhibiting the invasion of the mesenteric nodes of non-immune animals. This vaccine was on field trial in 1957.

Chemotherapy of the disease has hitherto been empirical, largely because no simple screening procedures have been available for determining the activity of new drugs. Recently a method of infecting guinea-pigs with the lungworm parasites of various domestic animals has been devised, based on techniques described for *Metastrongylus apri* at Liverpool. Many synthetic compounds have been examined in infected guinea-pigs, and one, cyanacethydrazide [6], was found to have a specific action against all the lungworms so far examined, when administered orally or by injection. The examination of this effect has been facilitated by the technique of intubating cattle or sheep and collecting in a small polythene bag any exudate or worms passing up the trachea [23] (Figures 4, 6). An intensive study of the drug in artificial and natural infestations of farm animals confirmed the laboratory findings for this new anthelmintic, which was employed extensively in the field during 1957.

Cyanacethydrazide in some way disorientates any lungworms and larvae present in the airways at the time of treatment, causing them to be carried out and swallowed with the bronchial exudate, which is continuously being removed by the ciliated epithelial cells of the respiratory tract. The adult worms and any fifth-stage larvae so removed are digested and destroyed. First-stage larvae survive, but the presence of phenothiazine in the bowel and faeces will prevent their further development. In any scheme for the control of pasture infestation, phenothiazine dosing at the time of cyanacethydrazide therapy should be considered, as the removal of egg-bearing female worms causes, for one or two days, a surge in the larval output of treated animals.

Timing of treatment is important. Naturally affected animals, provided that they survive the earlier acute stages of the disease, begin to expel their lungworm burden about the 45th day after infection, at which stage anthelmintic therapy is relatively valueless; the correct time to start treatment is as soon as the condition becomes apparent, i.e. 20–30 days previously. In some cases this will be before any substantial quantity of worms reach the airways, and therefore repeat treatment is advisable. Mass treatment is necessary because of the nature of outbreaks, and all animals in a group should receive the drug at the same time; even so, individual cases may merit special treatment, especially where marked bronchial exudation is impeding the movement of worms from the respiratory tract.

The margin of safety of cyanacethydrazide is satisfactory. In a large-scale toxicity trial on dairy and store cattle, doses on three successive days up to 10% greater than those required for therapeutic effect were not harmful. As dosage is based on body weight and not on age groups, no serious harm should follow from treatment provided the maximum doses are not exceeded.

Use of this remarkable new anthelmintic does not remove the necessity for measures against any of the concomitant parasitic or bacterial conditions so frequently present, for the drug has no effect on the pathological changes accompanying the disease, either in the lung or in the general condition of the animal. Management of affected animals must include shelter, especially at night, and extra food; indeed, the absence of such common-sense precautions may vitiate the treatment with cyanacethydrazide.

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BOOK REVIEWS

Agriculture in the British Economy. (Proceedings of Conference held at Brighton on 15–17 Nov. 1956.) Edited by Sir J. Scott Watson. Pp. 311, with line illustrations. Imperial Chemical Industries Ltd., Central Agricultural Control, London. 1957. 21s. net.

This publication contains the papers and discussion at a conference held at Brighton in November 1956. The title was "Agriculture in the British Economy," but the main emphasis was on the future—in particular the likely position of farming ten years from now.

The list of speakers was impressive and included a large number of the leaders in the industry. Unlike many similar conferences, this one allowed ample time for papers and comment, so that the discussion was detailed and authoritative. The proceedings should therefore prove a useful source book for speakers to farming audiences.

The opening session was largely devoted to the place of farming in the economy. As might be expected, the economists were by no means in full agreement. Professor Robinson forecast an increase in the standard of living of some 30% and an increase in food purchases of about 12%. To sustain the rise in output required, far more raw materials would have to be imported, but a corresponding increase in exports to pay for them is unlikely. For this reason British farming is likely to play a vital role in closing the gap. Professor Nash, on the contrary, pointed out that, as assistance to farming in one form or another was worth some £300 million, a saving of 5s. in the £ could be made by substituting imports from the cheapest source for home production. In some cases (e.g. cheese or condensed milk) it is doubtful whether the products pay for the imported feedingstuffs used to produce them. The fostering of uneconomic output could thus be a national liability and a misuse of resources.

Some interesting comments on trends were given by Drs. Raeburn and Wright in the discussion. The latter forecast a further shift towards the "American" type of diet rich in protein. He also showed how much of the improvement in recent years has been due to a levelling up of the former poorer classes.

The second session dealt with individual enterprises. Sir Peter Greenwell gave quite an optimistic forecast for cash crops and suggested that, with improved methods, home production was in a better position to stand competition from imports than was the case pre-war. With higher yields, the present output of potatoes, sugar beet and wheat could be obtained from a smaller acreage, leaving room for a substantial increase in feeding barley. The future for milk was largely concerned with the disposal of the so-called "surplus." Mr. Trehane forecast an increase of 1¼% per year or 250 million gallons by 1965. About half of this could be sold liquid and the rest absorbed in the manufacturing market. The inevitable fall in farm prices would have to be met by more efficient production, particularly in feeding.

The third session was devoted to means of achievement—chiefly man-power and capital. Mr. Currie made a plea for the extension of work study methods to farming and Dr. Price and Mr. Chambers dealt with capital and finance.

There is no space here to mention all the papers or to do justice to the wide range of topics discussed. Throughout the sessions two themes kept recurring—the instability of an industry where almost the whole profit is dependent on Government assistance and the necessity to reduce costs. As Mr. Messer and others pointed out, such assistance as is given should be devoted to permanent improvement rather than to short-term palliatives. On the whole, there was much quiet optimism that improved techniques could, in time, solve many of the problems of the industry. No one can expect a conference of this type to be either consistent in its recommendations or even accurate in its predictions, for there are too many uncertainties. But as an intelligent piece of crystal-gazing the contributions are most stimulating, and the directors of I.C.I. are to be congratulated on their enterprise in staging this gathering.

F. STURROCK

Types of Rural Economy. (Studies in World Agriculture.) R. Dumont. First English edition, translated from French by D. Magnin. Pp. xii + 556, with line illustrations. Methuen & Co. Ltd., London. 1957. 45s. net.

Professor Dumont has undertaken an ambitious task—the comparison of the rural economy of 15 different regions, in the tropics, western and eastern Europe, and North Africa. There is a French tradition of taking such grandly sweeping views of agriculture, established by Gourou and by the late A. Chevalier in some of his works. The only comparable English book is Sir John Russell's *World Population and World Food Supplies.*

Professor Dumont is a worthy successor to these illustrious forerunners in the diligence of his travels and his scrupulous collection of facts. His method of presentation, however, is unusual. After a slight sketch of the agricultural background in each region, two or three individual farms or villages are described in some detail as case studies to illustrate farming practice. While this may give a vivid impression to the general reader, it will perhaps be less useful to the student of agriculture. We have been so well supplied in recent years with sample surveys based on statistical principles that we have become accustomed to being given figures that we can confidently use as representative. Since the figures presented here cannot be used in this way, there is a risk that the mass of detail described may merely confuse the reader's picture. Nevertheless, so far as can be checked for regions known to the reviewer, the data given are correct and accurate and provide a fair picture of the rural economy of each area. This lends credibility to the very interesting account of three countries behind the "Iron Curtain," which gives a slightly more favourable impression of agriculture under Communist government than is probably generally current in the West.

The author does not spare his criticisms of official policy, especially in French territories, both metropolitan and overseas, and this may give a rather unfair impression, since he does not deal with the overseas possessions of other powers except the Belgian Congo, where he does not find quite so much to criticize. The great bogy at which he tilts is "Malthusianism"—in the French sense of restriction of production, or at least of a lack of stimulus to its increase. The space devoted to general arguments, however, in the closing chapter and elsewhere does not allow the author to develop his themes sufficiently for all of them to carry full conviction.

The book nevertheless serves its author's and the general purpose well in reminding us of the imperative need for better production and distribution of scarce foods and of the irrational inefficiencies that still occur in all farming systems, from the most primitive to the most advanced.

G. B. MASEFIELD

Progress in the Physiology of Farm Animals. Edited by J. Hammond. Vol. III. Pp. v + 300, with a 28-page index to volumes I–III and line and half-tone illustrations. Butterworths Scientific Publications, London. 1957. 50s. net.

The third and final volume in this series continues the subject of reproduction and concludes with a three-chapter review of milk production.

S. A. Asdell, in a commendably brief way, deals with the functions of the individual reproductive hormones and shows how these numerous substances regulate the oestrous cycle. J. A. Laing provides an excellent review on female fertility which contains much information of value to non-physiologists interested in animal breeding; normal fertility of farm animals is discussed, also factors affecting fertilization of ova and maintenance of the embryo during pregnancy; brief mention is made of work done to improve ovulation and of the main causes of depressions of fertility. T. J. Robinson has attempted in 110 pages to cover all aspects of pregnancy, some in considerable detail, and there is, not unnaturally, some duplication of information contained in other chapters; considerable space is devoted to the morphological aspects of pregnancy, and this is followed by sections on pre-natal nutrition, growth of the uterus, foetus and placenta, prenatal mortality, pregnancy hormones, pregnancy diagnosis, parturition and problems of pregnancy.

A. T. Cowie discusses the anatomy of the udder, growth of the mammary gland, milk secretion and milk removal. F. H. Dodd's contribution on factors affecting the rate of secretion of milk and lactation yields is less purely physiological and gives much evidence collected from farm operations. A chapter on milk composition by I. Johansson and O. Claesson seems out of place in this volume: most of the information on the constituents of normal milk can be found in standard textbooks, and it is disappointing that so little is said about the present-day problem of low solids-not-fat content of milk.

As in the previous volumes, all the articles have adequate bibliographies. A regrettable feature is that the chapter on male fertility could not be included in this volume: it is still in preparation, and must be obtained from the publishers later.

W. S. FERGUSON

ABSTRACTS

CROP PRODUCTION

The effect of nitrogen on cereal yields. E. R. Bullen and W. J. Lessells, *J. Agric. Sci.*, 1957, **49**, 319–28.

The results of 270 experiments on wheat barley and oats in England and Wales in the last decade are summarized. Recommended dressings for highly responsive varieties of winter wheat (Hybrid 46, Cappelle Desprez, Nord Desprez, Bersée, Atle) and spring barley (Kenia, Proctor, Herta, Freja) is 0·6–0·7 cwt. N/acre, producing a net return (1956 prices) of £10 per acre; for other varieties of winter wheat and spring barley and for spring wheat the recommended dressing of 0·5 cwt. N/acre produces a net return of £5 per acre; for spring oats the recommended dressing of 0·3 cwt. N/acre produces a net return of 30s. per acre. In places with a very high rainfall the recommended nitrogen rates are lower. Where the rainfall in the first three months after sowing was less than 10 in., the average response of winter wheat to autumn nitrogen equalled that to spring dressing. S.F.A.

Note on the effect of stubble and straw residue on the availability of nitrogen. W. S. Ferguson, *Canad. J. Soil Sci.*, 1957, **37**, 145–6.

Experiments were carried out to estimate the quantity of fertilizer nitrogen required to overcome the depressing effect of cereal crop residues on nitrogen availability in soils. Three rates of nitrogen (0, 30, 60 lb./acre) factorially combined with four rates of straw (500, 1500, 3000, 4500 lb./acre) were tested on two soil types. Results showed that, under the conditions of these experiments, cereal residues were not as important in influencing the availability of nitrogen in soil as had been expected from other reports on the subject. E.M.A.

Relationship of nitrate accumulation to yield response of wheat in some Saskatchewan soils. F. D. Cook, F. G. Warder and D. L. Doughty, *Canad. J. Soil Sci.*, 1957, **37**, 84–8.

A laboratory determination of nitrate accumulation in Saskatchewan soils has been investigated as a method for evaluating the possible response of wheat to nitrogenous fertilizers. It is considered that a response to nitrogen can be expected when the nitrate accumulation value is under 50 p.p.m for soils cropped the previous season and 40 p.p.m for fallowed soils. E.M.A.

The value of mid-season top-dressing of nitrogen fertilizer for main-crop potatoes. G. W. Cooke, F. V. Widdowson and J. C. Wilcox, *J. agric. Sci.*, 1957, **49**, 81–7.

Nitrogen applied wholly to the seedbed (as ammonium sulphate) was compared with equivalent dressings applied partly to the seedbed (as ammonium sulphate) and partly as a top-dressing ('Nitro-Chalk') just before the final ridges were set up. Splitting the dressing was inferior to applying it all to the seedbed on medium and heavy soils; there was a very slight advantage from splitting the dressing in about half the experiments on light soils. Top-dressings did not prolong growth at centres where the crops were considered to have died prematurely. When the nitrogen was all applied to the seedbed, dressings of 0·5 and 1·0 cwt. N/acre consistently increased yields; 1·5 cwt./acre seedbed N increased yields further in some experiments on light soils but depressed yields at most heavy-land centres. S.F.A.

The influence of straw and straw-fertilizer composts on the uptake of fertilizer phosphorus by plants. W. H. Fuller and D. R. Nielsen, *Proc. Soil Sci. Soc. Amer.*, 1957, **21**, 278–82.

The value of the widespread practice in the S.W. United States of applying inorganic phosphorus solutions to crop residues before ploughing into the soils was investigated in pot trials. By using radioactive phosphorus it was shown that more phosphorus was taken up by rye-grass from wheat straw compost than from wheat straw, but phosphorus-free extracted oat straw treated with KH_2PO_4 supplied more phosphorus than a similarly treated oat straw compost. It is thought that the degree of decomposition of the residue may affect these results. There appeared no advantage in adding liquid phosphoric acid to crop residues before incorporating them into the soil over the practice of applying fertilizer and crop residue independently. E.M.A.

The effect of leys on soil fertility. 1. The effect of management of clover aftermath on the yield of the succeeding wheat crop. F. Hanley, W. J. Ridgman and M. G. Barker, *J. agric. Sci.*, 1957, **49**, 251–7.

Broad red clover was cut for hay in June and the aftermath subjected to one of five treatments: (1) ploughed immediately the hay had been removed, then bastard fallowed; (2) aftermath cut at the full-flower stage and removed; (3) aftermath grazed by sheep at the full-flower stage; (4) aftermath ploughed in at the full-flower stage; (5) aftermath allowed to grow to the ripe-seed stage, than cut and removed. Plots receiving treatments (2), (3) and (4) were all ploughed the same day, usually at the end of August; the plots receiving treatment (5) were ploughed in October. Wheat was then sown at the same time on all plots. The experiments were carried out on different sites for six years. In plots receiving treatments (2)–(4), which all had the clover growing for the same length of time, there was little difference in wheat grain yield, but the percentage of nitrogen in the grain was higher after ploughing-in or grazing the clover than after cutting for hay. In treatments (1), (2) and (5) the aerial portion of the crop was removed, but dates of ploughing and the length of growing period of the clover differed. Fallowing generally gave better braird, more tillers, better early spring growth and greater straw yield than the other treatments, but the grain yield was generally less than that of the hay treatment. Taking the second crop for seed led to a rough seedbed, a poorer braird, fewer tillers, less straw, and generally lower yields than from plots that had been cut for hay. S.F.A.

Conjoint effects of reseeding, fertilization and mole-draining on vegetation and output of permanent meadowland in County Antrim. P. A. Linehan and J. Lowe, *Res. exp. Rec. Minist. Agric. N. Ire.*, 1954 (pub. 1956), **4**, 8–15.

A field of 8¾ acres was divided into 4 plots which received the following treatments: (1) reseeded, mole-drained and fertilized annually; (2) old sward mole-drained and fertilized annually; (3) reseeded, not mole-drained and not fertilized; and (4) old sward not mole-drained and not fertilized. Dry matter yields for the five years 1950–4 showed no benefit from reseeding, but mole-draining and fertilizing gave yields 159% of the untreated control. The botanical composition of the old sward did not change materially over the period. Mole-draining and fertilizing the old sward increased Yorkshire fog and meadow fescue. On the unfertilized, undrained, reseeded plot timothy and Yorkshire fog increased. The reseeded and fertilized plot became and remained timothy dominant. S.F.A.

The renovation of downland permanent pasture. 2. Herbicidal treatment, surface cultivation and seeding. M. J. T. Norman and J. O. Green, *J. Brit. Grassl. Soc.*, 1957, **12**, 74–80.

In spite of initial differences in the establishment of S.24 perennial ryegrass and S.100 white clover, pretreatment with 2,4-D (amine) had no effect on the cover of sown and unsown perennial ryegrass and white clover two years later. The increase in dry-matter yield resulting from renovation was only about 10%, but the increase due to grazing management and adequate NPK manuring may have been nearly 100%. The ingress of undesirable creeping grasses could be checked by close grazing with either sheep or cattle soon after spraying. S.F.A.

CROP PROTECTION

Crabs as pests of rice on tidal swamps. H. D. Jordan, *Emp. J. exp. Agric.*, 1957, **25**, 197–206.

At the West African Rice Research Station, crabs (especially *Sesarma huzardi*) caused serious damage to rice seedlings growing on tidal lands. Three acres of selected lines were destroyed in 1951 within two weeks of planting. Technical BHC was found to be repellent in baits of grated cassava or rice bran and groundnut cake, but it acted more efficiently as a contact poison. Lindane acted more quickly at equivalent concentrations. Spraying with 10% technical BHC (containing 6·5% γ-isomer) was adopted as an effective routine measure, and even when crabs were not killed they quickly stopped feeding. Mud skippers were unaffected by the spray. For the protection of individual plants of selections and hybrids, technical BHC was used at ½ oz./sq. yd. D.L.

Isolation and synthesis of an insect resistance factor from corn plants. E. E. Smissman, J. B. LaPidus and S. D. Beck, *J. Amer. chem. Soc.*, 1957, **79**, 4697–8.

The compound 6-methoxybenzoxazolinone has been isolated from dried maize plants at the University of Wisconsin as one of three chemical factors rendering certain maize strains resistant to attack by the European corn borer (*Pyrausta nubilalis*). The compound was known to exist, and had previously been described as "Factor A." (It is of related interest that Finnish workers in 1955 identified a benzoxazolinone as an anti-fusarium factor in rye seedlings.) D.L.

The systemic action of lindane in alfalfa upon the meadow spittlebug. C. S. Koehler and G. G. Gyrisco, *J. econ. Ent.*, 1957, **50**, 346–7.

The translocation of lindane in the lucerne plant is believed to be the reason for the high specificity the insecticide has shown against sap-feeding nymphs of the meadow spittlebug, *Philaenus leucophthalmus*. The supposition was tested at Cornell University by placing lucerne plants, each having two long stems, in boxes so that one stem could be sprayed without contaminating the other. Five days after treatment with emulsifiable lindane at concentrations of 0·05–0·25% active ingredient, the sprayed stem was cut off and test insects were placed on the remaining, untreated stem. Kills of 37·5% were given within 30 hours by the highest concentration and 17·5% by the lowest concentration. Systemic action from soil applications of the insecticide was also demonstrated in pot experiments in which great care was taken to avoid the possibility of fumigant action. Six days after soil treatment with 20 ml. of the same lindane concentrations, spittlebug nymphs were released on the plants. Total kills were given by the 0·25% material 20 hours later, and by 0·1% after 50 hours. Reinfestation experiments with soil-treated plants showed that up to 97·5% control was still possible six weeks after treatment. D.L.

The systemic behavior of *O,O*-diethyl *S*-2-(diethylamino) ethyl phosphorothiolate and its salts. R. L. Metcalf, E. M. Stafford, T. R. Fukuto and R. B. March, *J. econ. Ent.*, 1957, **50**, 205–10.

The systemic insecticidal behaviour of the thiono- and thiol-isomers of *O,O*-diethyl *S*-2-(diethylamino) ethyl phosphorothiolate was evaluated in cotton and lemon plants by using labelled phosphorus. The rates of uptake and translocation of the thiol-isomer base (recently given the common name of "amiton") and its oxalate were compared after having been applied to the stems and in solution to the roots. From root applications to cotton, both chemicals accumulated in the leaves at about equal rates; from stem applications, the thiol-isomer base accumulated ten times more rapidly than the salt, and this was considered to be due to the preferential influence of lipoid solubility. A similar relationship was found in the case of lemon. The distribution of the thiol-isomer oxalate in citrus fruits was measured after trees had been sprayed at a rate of 40 g./100 gal. water. No residue was detected in the pulp of oranges or lemons, but in the peel of those fruits amounts of from 0·08 to 0·32 p.p.m.

were present within four weeks of spraying. The LD-50 concentration of the thiol-isomer base to the two-spotted red spider mite *Tetranychus telarius* was established at 11 p.p.m. in cotton leaves after 24 hours. Amiton and amiton-oxalate were compared with demeton thiol-isomer as cotton seed treatments. The base accumulated in the cotyledons and leaves about 5–10 times more rapidly than the oxalate, and demeton thiol-isomer accumulated more rapidly than the base. Amiton probably acts as an endolytic systemic toxicant, for there was no evidence that it or its oxalate was metabolized to other toxicants in the juices of lemon or cotton leaves. D.L.

Chemically induced resistance to stem rust of wheat by derivatives of Acti-dione. R. G. Hacker and J. R. Vaughn, *Plant Dis. Reptr*, 1957, **41**, 442–6.

In greenhouse and field trials with Acti-dione derivatives against wheat stem rust (*Puccinia graminis tritici*), the semicarbazone analogue of cycloheximide gave complete protection at 200 p.p.m. for 38 days. The absence of pustules compared with 65% average infection in control plots. The compound was almost as active at 100 p.p.m., but the acetate of cycloheximide was not so effective. The mode of action is not understood, but the antibiotic is postulated to impart a chemical resistance to the establishment of the rust fungus in the host tissues. It is also probable that the inhibition is brought about by the extreme persistence of the compound. Both derivatives at 200 p.p.m. caused only slight foliar spotting, with no adverse effects on yield or germination. D.L.

Attempts in the chemotherapy of virus-infected glasshouse crops. R. Howles, *Plant Pathology*, 1957, **6**, 46–8.

In varied trials with chemicals against plant viruses at the Glasshouse Crops Research Institute, Littlehampton, most materials tested were of no value. A single application of 0·2% 2,4-D showed some degree of control over tomato aspermy virus on chrysanthemums, whether applied to the foliage or to the roots. There was some damage to the shoot system, but cuttings taken from the plants were normal. Proceeding from these observations, eleven chlorinated phenoxyacetic acids and salts were examined; although the majority had little effect, two were promising. 3-Chlorophenoxyacetic acid caused no damage from a single application at 0·013% and apparently freed 4 plants in 20 from virus. The ammonium salt of 2,4-D at the same concentration gave the same degree of control with slight phytotoxicity; with two applications damage was severe, but more than half the plants were freed of virus. Malachite green at 0·00625% retarded the multiplication of aucuba mosaic virus and potato virus X in tomato plants, and at 0·025% delayed the onset of symptoms when applied to the soil at a rate of 100 ml./pot on each of 5 days before inoculation. Half-saturated 2-chlorophenoxyacetic acid also delayed the appearance of symptoms in the latter test. 2-Chlorophenoxyacetic acid was also successful in freeing tomato seed from tobacco mosaic virus, giving normal germination and seedlings. The seed was treated at a rate of 2 g./16 ml. saturated acid for 6 days. Mosaic-infested lettuce seed steeped in a 0·02% solution of the acid for 2 days produced infected seedlings. D.L.

Combating plant virus diseases with a new antiviral agent, Cytovirin. R. A. Gray, *Plant Dis. Reptr*, 1957, **41**, 576–8.

Local lesion formation by southern bean mosaic virus on Pinto beans and by tobacco mosaic virus on *Nicotiana rustica* was completely prevented when plants were sprayed with a culture filtrate of an unidentified *Streptomyces* sp. at a dilution of 1 : 3 with water. A pure crystalline product, Cytovirin, was later prepared; it controlled lesion formation by southern bean mosaic virus at 1 p.p.m. and by tobacco mosaic virus at 0·5 p.p.m. Systemic infections by these viruses were controlled by the use of the crude filtrate, sprayed at 100 p.p.m. two hours after inoculation and again 12 days later. Cytovirin would not eradicate the viruses if they were thoroughly established throughout the plant. D.L.

Studies on breeding red clover for resistance to stem nematodes. S. Bingefors, *Växtodling*, 1957, No. 8, 123 pp.

The stem nematode, *Ditylenchus dipsaci*, is widespread on red clover in Sweden. Although it may be decreased in numbers by crop rotation, it is difficult to allow a sufficient time-interval between red-clover crops. Chemical control has not given useful results. Dependence is therefore placed on the use of nematode-resistant clover varieties. Resistant semi-late varieties are widely used, and now winter-hardy varieties have been developed. The initial selection of seedlings for resistance, from inoculation of the cotyledons with suspensions of nematodes, was carried out in the laboratory, and the degree of infestation assessed after about 10 weeks. The work provided some information on race differentiation in the species. For example, the red-clover race did not cause field damage to lucerne, but in laboratory tests the nematode penetrated both red clover and lucerne plants. The nematodes did not reproduce in lucerne, nor did the lucerne race reproduce in red clover. Alsike clover was attacked by the red-clover race, but no damage resulted. Merkur was the variety found to be most resistant, and its resistance was not decreased by seed production for 3 or 4 generations. It was sometimes possible to improve the resistance of a susceptible variety by selection for 2 generations, although usually a longer period of selection was required before the same degree of resistance stability as that of Merkur was reached. Doubling of the chromosome number did not usually influence resistance. In breeding, crosses between resistant and susceptible varieties gave progeny whose resistance was medial between that of the parents. Crosses between resistant varieties produced not more than 95% resistant progeny, and it was apparent that a few major genes were mainly responsible for the inheritance of resistance. Nematode penetration was not prevented in resistant plants, but the eelworms were unable to reproduce and soon died out. D.L.

Clover tolerance. F. B. Thompson, *9th N.Z. Weed Cont. Conf.*, 1956, 89.

Replicated trials were carried out in 1954–5 on pure, ungrazed stands of spring-sown and autumn-sown white clover (*Trifolium repens*), subterranean clover (*T. subterraneum*) and red clover (*T. pratense*). Derivatives of phenoxyacetic, phenoxybutyric and phenoxypropionic acids were applied at 1 lb. a.e. per acre, except for some of the 2,4,5-T treatments which were applied at ½ lb. a.e. per acre, to seedling (2–3 trifoliate leaves) and mature clovers. Generally the butyric acid derivatives were less toxic than the acetic acid derivatives, and the propionic acid derivatives more toxic than the other two. The difference between the acetic acid and butyric acid derivatives was most marked with red clover and least with subterranean clover. The sodium salt of MCPB caused less damage than any other material tested. The butoxy ethanol ester of 2,4-DB gave less damage to spring-sown seedlings and mature red clovers than the butoxy ethanol ester of MCPB, but in a previous field trial on mature red clover in a pasture with the butyl esters of both there was more damage with the 2,4-DB ester than with the MCPB ester. 2,4,5-T and 2,4,5-TP esters reduced the stand of autumn-sown seedling clovers, the propionic more on the white, and the acetic more on the red. The addition of $\frac{1}{2}$ lb. amino triazole per acre to 2,4,5-T at $\frac{1}{2}$ lb. a.e. per acre reduced the toxicity of 2,4,5-T on subterranean clovers but made no difference on red and white clovers. All autumn treatments to seedling clovers were more severe than similar treatments on spring-sown seedling clovers. The relative order of toxicity for the different derivatives was the same for both seedling and mature clovers, although generally the mature clovers showed slightly more damage. R.A.U.

The selective herbicidal properties of several variously substituted phenoxyalkylcarboxylic acids. W. C. Shaw and W. A. Gentner, *Weeds*, 1957, **5**, 75.

Preliminary tests were carried out on various ring substitutions of phenoxyacetic, alpha and beta phenoxypropionic and alpha and gamma phenoxybutyric acids to investigate the relationship between their chemical structure and selective herbicidal activity. The pure acids were applied pre- and post-emergence, under greenhouse and field conditions, at several rates to a comprehensive range of crops and weed species. Data support the theory that plants with the proper enzymatic constitution may degrade omega phenoxyalkylcarboxylic acids with an even number of carbon atoms in the side chain to herbicidally active derivatives, while those with an odd number of carbon atoms in the side chain are degraded to herbicidally inactive products. The phenoxyacetic, alpha phenoxypropionic and alpha phenoxybutyric acids all showed fairly similar ranges of selectivity and specificity, but the phenoxyacetic acids exhibited a slightly higher average level of inhibitory activity than the other two. The alpha phenoxybutyric acids showed lower activity on Brassica spp. than the phenoxyacetic and alpha phenoxypropionic acids. The beta phenoxypropionic acids, with an odd number of C atoms in their side chain, were the least active of the compounds tested, and only exhibited very slight localized, contact phytotoxicity. The gamma phenoxybutyric acids, with an even number of C atoms in their side chain, possessed herbicidal specificity in crops sensitive to phenoxyacetic and alpha phenoxypropionic acids, and showed promise as herbicides in many legume and legume associations and on tolerant crops grown near phenoxyacetic and alpha phenoxypropionic sensitive crops. Ring substitution had a great effect on the herbicidal activity of the phenoxyalkylcarboxylic acids, and higher general activity was usually associated with the 2,4-dichloro, 2-methyl-4-chloro and 2,4,5-trichloro substitutions than with the 3,4-dichloro and 4-chloro derivatives. The first three substitutions of the alpha phenoxybutyric, phenoxyacetic and alpha phenoxypropionic acids showed higher activity on legumes than on oats, corn and brassicae; this was most apparent with alpha phenoxybutyric acids. The gamma phenoxybutyric acids showed a marked difference as the 3,4-dichloro and 4-chloro derivatives were more active on legumes than the 2,4-dichloro, 2-methyl-4-chloro and 2,4,5-trichloro substitutions. It was stressed that if a chemical is to be beta-oxidized within a plant it must possess physical properties to enhance its penetration into plants, structural properties to facilitate its translocation to the places of activity and to enable adsorption and correct orientation at the surface of the enzymes, a side chain structure that is subject to beta oxidation, and a ring structure that will not interfere with beta oxidation. R.A.U.

Dowpon—Lalang and grass eradicant. Anon., *R.R.I. Plant. Bull.*, 1957, No. 29, 27.

In 1956 Dowpon (dalapon) showed great promise as a grass eradicant under Malayan conditions. Recent trials have shown that the best rate to control lalang (*Imperata cylindrica*), bamboo grass (*Ischaemum muticum*) and other grass weeds in rubber estates is 10 lb., or a maximum of 15 lb., of Dowpon in 100 gal. water, plus wetting agent, per acre. Four months after treatment with Dowpon at 10 lb./acre unslashed lalang showed a 5–10% regeneration, whereas 40 lb. was necessary to give an equivalent control of slashed lalang. The residual activity of Dowpon does not prevent recolonization by Siam weed (*Eupatorium odoratum*) and other broad-leaved plants such as *Borreria latifolia.* From experimental results it was thought that an application of 15 lb. sodium arsenite to vigorous sheet lalang before the Dowpon spraying might weaken the original stand, and 18 in. regrowths might be controlled by 10 lb. of Dowpon per acre. Young rubber may be damaged by Dowpon, and probably not more than 5 lb./acre should be used in very young nurseries. LCB1320 is a susceptible clone, Tjir 1 shows intermediate susceptibility, and PR107 and RRIM501 are two of the least sensitive clones. Repeat applications at two-monthly intervals of Dowpon at 2–5 lb./acre may give selective grass control in leguminous cover crops, such as *Pueraria phaseoloides*, but a single application of 10–20 lb./acre killed the legume. Dowpon has also been claimed to give good bracken control at 15–20 lb./acre. R.A.U.

Chemical weeding and thinning when using pelletized vegetable seed. V. L. Guzman, *Weeds*, 1957, **5**, 227–36.

Pelletized seeds of lettuce, cabbage and pepper were planted 2·5–4·5 in. apart, and CIPC at 10 lb./acre (in lettuce), CDEC at 4–8 lb./acre (in cabbage) and Oktone at 10 lb./acre (in pepper) were used as pre-emergence weedkillers. Chemical thinning using a herbicide mixed with oil 2–3 weeks after emergence was compared with mechanical thinning by a tractor equipped with either mechanically or electrically operated valves. Chemical thinning gave more efficient weed control, and pre-emergence application of the herbicides facilitated chemical thinning when using pelletized seed. Chemical thinning was also more effective when seedlings were 3·5 and 4·5 in. apart in the row than at closer spacings. E.M.A.

ANIMAL HUSBANDRY

Studies on the extension of the grazing season. 1. The evaluation of selected strains of some grass species for winter grazing. 2. Pasture management for foggage production. J. L. Corbett, *J. Brit. Grassl. Soc.*, 1957, **12**, 81–96, 187–96.

Plots of ryegrass, cocksfoot or timothy at the Rowett Research Institute were strip-grazed at various times during three successive winters by store cattle maintained almost entirely on foggage; cocksfoot was the most suitable species. A small plot trial on cocksfoot-dominant sward cut on 1.12.54, 1.2.55 and 1.4.55 showed that optimum yields could be obtained by closing fields in mid-August and applying 3 cwt. 'Nitro-Chalk' per acre; potash had no effect. Closing in early September gave smaller yields of a higher protein foggage and the sward was less cocksfoot-dominant. S.F.A.

Artificial rearing of calves on pasture. T. R. Preston, *J. Brit. Grassl. Soc.*, 1957, **12**, 178–86.

In a preliminary experiment at Durham University in 1953 spring-born Ayrshire calves reared on pasture from birth made similar weight gains to control animals reared conventionally indoors. In 1954 in a more extensive experiment, however, the outside-reared calves gained weight at a significantly slower rate than their pair mates, and six of the ten calves outside lost weight in August or September. The nutritive value of the grass was high throughout, but in wet and cold weather the amounts of total digestible nutrients consumed fell below the theoretical requirements of the calves outside. Rotational grazing with heavy stocking (5 calves per acre) resulted in a heavy worm burden which coincided with the wet weather in August. S.F.A.

The use of autumn grass for weaned calves. I. A. Nicholson and R. G. C. Smith, *Proc. Brit. Soc. Anim. Prod.*, 1957, 33–40.

Performances of two groups of weaned calves, one housed and fed in courts immediately after weaning while the other remained on grass for several weeks prior to court feeding, were compared in three trials extending over three seasons. In 1952 the grazing season was one month, but in 1953 and 1954 this was extended to two months. Setback in live-weight increases occurred when the grazing calves were moved into the courts, and this fall could not be counteracted even by feeding a hay supplement to calves a few days prior to moving indoors. In all years the group receiving continuous court feeding from weaning had an advantage in weight over the others at the end of the winter period; nevertheless the information presented shows that autumn grass can make a contribution to calf feeding with a considerable saving of the more expensive feeds for indoor rations. E.M.A.

Oestrogens in lamb and mutton production. T. R. Preston and I. Gee, *Proc. Brit. Soc. Anim. Prod.*, 1957, 41–8.

Suckling one-month-old lambs, implanted subcutaneously in the ear with a 15 mg. pellet of hexoestrol, reached a slaughter weight of 80 lb. twelve days earlier than controls. There was no significant difference in dressing percentage or in carcass grading between treated and untreated animals. When eight-month-old weaned lambs and six-year-old draft ewes were implanted in each ear with 7·5 mg. pellets of hexoestrol, significant increases in weight over control animals were obtained accompanied by improvement in carcass quality. E.M.A.

The treatment of contagious footrot in sheep by the topical application of a 'Cetavlon' tincture. B. A. Forsyth, *Aust. Vet. J.*, 1957, **33**, 157–61.

Footrot in sheep has increased greatly during the last few wet years in the southern states of Australia, and the author investigated its specific treatment. Using natural cases of footrot, a screening test of seven antiseptics was carried out. The affected feet were pared thoroughly, an antiseptic was applied and the foot re-examined after 2 or 3 weeks. By far the most effective treatment was a 20% tincture of Cetavlon, which cured 21 out of 24 feet (87·5%). A 20% aqueous solution of Cetavlon cured 11 out of 15 feet (73%), and the next most effective substance was hexachlorophene as a 10% tincture, which cured 4 out of 7 feet (57%). Hibitane, aqueous sodium pentachlorphenate, and sodium orthophenylphenate tincture cured less than 50% of affected feet. Further work carried out on farms with a troublesome incidence of footrot was done with Cetavlon in comparison with chloramphenicol and formalin with the following results: In one trial 20% aqueous Cetavlon cured 73, 83 and 95% of feet after one, two and three treatments respectively; corresponding figures for 20% Cetavlon tincture were 68, 94 and 99%. In three other trials the cure with 20% Cetavlon tincture ranged from 66·6 to 82% after one treatment and from 87·5 to 97% after two treatments; corresponding figures for chloramphenicol 10% tincture were 67·8–83% and 86·6–95%. In one trial, cures from one and two treatments were 72·1 and 90·5% with Cetavlon 30% aqueous solution and 61·1 and 81·2% with 10% formalin. On another property a comparison was made of a 10% formalin foot-bath with the same preceded by the application of a 20% Cetavlon foot-paint to pared feet, which were allowed to dry for 1 hour before the formalin foot-bath. The combined treatment cured 69% of 93 feet, whereas the simple formalin foot-bath cured only 35% of 103 feet. [Presumably the formalin foot-bath added nothing to the success in the combined treatment group.] Comparison showed that a 20% tincture of Cetavlon is equivalent to a 10% tincture of chloramphenicol: a single treatment with the former cured 72% of 282 feet as compared with a cure rate of 73% of 134 feet treated with chloramphenicol: after two treatments the cure rate had risen to 91 and 93% respectively. It is concluded that 20% is the optimal concentration of Cetavlon which in tincture form is indicated for footrot in sheep. The cost of its use is about one-tenth that of 10% chloramphenicol tincture. J.T.E.

A study of subclinical worm infestation in sheep. Part 3. The effect on wool production. C. R. W. Spedding and T. H. Brown, *J. agric. Sci.*, 1957, **49**, 229–33.

Worm-free sheep produced 12·0–38·8% higher total fleece weights and 17·2–43·2% more clean dry wool per unit area than infected sheep; none of the infestations was higher than occurs naturally in the field. The effect on wool growth is intensified when the animal is infected at an early age, when the level of nutrition is low, and when the infestation is in winter. S.F.A.

Systemic effect of Dipterex on the bed bug and Gulf Coast tick when administered to rabbits. T. R. Adkins and F. S. Arant, *J. econ. Ent.*, 1957, **50**, 166–8.

High kills of fifth-instar bed bugs (*Cimex lenticularis*) and of nymphs of the Gulf Coast tick (*Amblyomma maculatum*), feeding externally on rabbits, were obtained by the internal administration of Dipterex (*O,O'*-dimethyl ester of 1-hydroxy-2,2,2-trichloroethyl-phosphoric acid). Dipterex at 40 mg./kg. gave 95·5% kill of bed bugs an hour after its administration by stomach tube to the host; after two hours the mortality rate increased to 100%. With tick nymphs, 100% mortality was caused by the administration of Dipterex at 100 mg./kg. At 50 mg./kg. there was 88·3% kill. LD50 levels, measured in quantities administered to the rabbits, were 14·0 mg./kg. for bed bugs and 36·0 mg./kg. for tick nymphs. D.L.

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